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RADIOLOGY IN THE ATOMIC AGE*

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MY FIRST words will be to thank you, Members of the American Roentgen Ray Society, for the honor bestowed upon me and to let you know how deeply I appreciate the distinction which this Society has conferred upon me. In the past this office has been held by leaders in American radiology who are responsible for the place our specialty holds in the radiology of the world. Many original contributions, usually presented first at the annual meeting of this Society, have come from these men. Hence it is with considerable apprehension that I assume the office with which you have honored me and offer a few remarks on the subject of "Radiology in the Atomic Age."

As Year One of the Atomic Age ends and Year Two begins it would seem reasonable to think that certain radiologists actually connected with the gigantic project of releasing atomic energy could best discuss this topic with us. But such is not the case due primarily to continued security restrictions upon these authorities and to the preoccupation of most of them with the data from the recent scientific tests at Bikini Atoll. However, one need not be a member of the Manhattan Engineer District Project nor of the select group of investigators at Bikini to appraise the effect of the first atomic explosion upon our specialty and to venture a short extrapolation into the near future.

The element uranium, which was to prove itself to be the "philosopher's stone" for the large scale transmutation of the elements in the 20th century, was discovered by Klaproth in 1789. It was 107 years later that the most important property of this element, namely, natural radioactivity, was determined, for it is characteristic of science that discoveries are rarely made except when people's minds are ready for them. After the discovery of the x-ray by Röntgen in 1895 every laboratory in the world took out its old Crookes tubes to produce roentgen rays. These tubes showed that the production of roentgen rays had the power of producing brilliant phosphorescence in the glass envelope of the tube and as roentgen rays appeared to emanate from these points of phosphorescence it was certain that some investigator would wonder if roentgen rays and phosphorescence were not in some way interrelated and attempt to produce darkening of a photographic

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plate by various phosphorescent substances. Thus it was that Henri Becquerel in February, 1896, discovered the natural radioactivity of uranium. His collaborators on the problem of determining the substance or substances in the uranium salts responsible for the emission of the radiation were Pierre and Marie Curie. Their laborious chemical analyses and fractional distillations finally culminated in the separation of two new elements, polonium and radium, and established the factual evidence and foundation for modern atomic physics.

There followed revolutionary years of experimental and theoretical advances by Rutherford, Einstein, the Joliot-Curies, Bohr, Chadwick, and the Americans, Urey and E. O. Lawrence. Half a century has passed since the Curies set off on the road which has led to the present discoveries, and so enormous is our modern technology that in the same length of time (five years) required for the Curies to extract a minute amount of pure radium from several tons of pitchblende the gigantic physical and engineering prowess of American industries, universities and laboratories has made atomic power available for war and peace.

Whatever direction the large-scale development and utilization of atomic energy takes it will influence medicine profoundly. As our mastery of atomic techniques develops, and the tremendous amount of research being carried out in the whole field of radioactivity finds wider clinical application, it is to the radiologist to whom members of the other medical specialties will turn for information.

The first point that I propose to discuss is the closer relationship that will exist between the radiologic physicist and the radiologist in the Atomic Age. Physics has now a more important part to play than ever before in medical research and even in the *daily treatment of the patient.* The collaboration of radiologist and radiologic physicist becomes now the sine qua non for correct and intelligent therapy with the new radioactive isotopes. An atomic pile in operation is a source of radioactive materials, many of which will be medically useful, far greater than any source previously known. As a direct result of the fission process of the uranium itself there are produced radioactive isotopes of elements located in the middle part of the periodic system of the elements. These radioactive substances can be purified chemically and used medically. In addition, an atomic pile is far more efficient in bombarding substances with neutrons than is a cyclotron, and as a result most elements can now be obtained in a radioactive form in far greater quantity than heretofore dreamed of. Since the radiations emitted by them are equivalent to the radiations emitted by radium, these substances will be used for medical purposes on a much greater scale than has been possible with radium. So if our reliance on the radiologic physicist has been great for the proper knowledge and physical data in the correct and intelligent utilization of radium how much greater will be our cooperation and alliance with the physicist in the Atomic Age! Mayneord,[1] writing on "Physics in Medicine" in a recent issue of the *British Medical Bulletin*, states the situation as follows: "The physicist must realize that however fascinating and important his more academic problems, his primary responsibility is to be useful, while on the part of the radiologist we ask for a more enlightened understanding of the importance of the physicist, not only in solving the technical day-to-day problems but also as a sprearhead of the attack on the fundamental biophysical problems of the structure of living material and its interaction with radiation."

To the faithful physicists, who as Associate Members of this Society have done so much to advance the physical foundations of radiology in this country, and through their service on the American Board of Radiology have painstakingly and laboriously raised the standards of the certified radiologists will fall the bulk of

the burdensome task of directing and guiding the medical application of each of the useful radioactive isotopes by the same thorough and time-consuming work that was required for radium dosage.

Another important consequence of atomic power development of the future will certainly be the health problems of such an industry, and the industrial hazards to health will be primarily radiological. By the very nature of things the industrial medicine of the atomic age will be radiological. In the development of methods of using atomic energy for military purposes the importance of health problems was early apparent and a health division of the atomic bomb project was organized under our colleague, Dr. Robert S. Stone. Medical men and radiologists, in particular, are extremely anxious to learn the results and detailed knowledge obtained by our colleagues, Dr. Stone, Dr. S. L. Warren, and a host of other investigators, in the work on this phase of the project. It seems we shall not learn of it fully at first-hand until that portion of the science involved in the beneficial uses of atomic energy can be divorced from the technology involved in making atomic weapons.

We do know, however, that work carried on by them was along three lines of inquiry: (1) provision of instruments and clinical tests to detect any evidence of dangerous exposure of the personnel; (2) research on the effects of radiation on persons, instruments, etc.; and (3) estimates of what shielding and safety measures must be incorporated in the design and plan of operation of the atomic energy plant. The important data collected in these three fields is certain to form the foundation of the radiological industrial medicine of the future. In the very near future, no doubt, the radiologist will have available for his use specialized detection equipment sufficiently reliable to permit its being correctly and successfully operated by technicians and others having little experience in the complexities of the electronic circuits involved. Professor Robley D. Evans recently wrote, "It should be possible to bring these instruments to nearly the same state of universal usefulness as a household radio." Thus a whole new branch of industrial medicine will open up for the certified radiologist, with him and his technicians operating in addition to the regular radiological equipment the many newly devised specialized types of detection devices and Geiger-Müller counters. The future developments in this field of occupational medicine, which I have called radiologic industrial medicine, will parallel, of course, the development and use of atomic energy for the production of controlled and usable power, and may not lie so remotely in the future as the time interval between the discovery of the x-ray and the construction of the modern Coolidge tube.

Another phase of our specialty destined to be greatly affected by the developments of the Atomic Age is that branch often spoken of as radiologic jurisprudence. A new cause of litigation arises out of the hazard incurred by personnel working with or near atomic piles, cyclotrons and their radioactive products. The two types of radiation hazard connected with an atomic pile or with a cyclotron are neutrons generated in the pile or during cyclotron bombardment and alpha particles, beta particles and gamma rays emitted by the radioactive products of a pile or cyclotron. Considering also that the amount of radioactive material to be handled, much of it by technical assistants of non-professional standing, is many times greater than has ever been encountered before, one can quickly realize the great expansion of medicolegal problems accompanying radiology in the Atomic Age.

Should the world be stupid enough to launch an atomic war, which would surely be the most deadly in history, what would be the military medicine of such a war? We can say it would be radiologic military medicine in that by far the greatest percentage of killed and wounded would be radiological casualties caused by the power-

ful and intense radiations of all types, and, perhaps, the medicomilitary measures against poison gas (anti-gas impregnated clothing and the gas mask) would be replaced in the atomic war by the inverse square law, lead and concrete protective devices and Geiger counters. But what do we or the Surgeon General know about the pathological physiology and treatment of radiation sickness and injury for those persons not killed outright but suffering from overwhelming or even moderate overexposure to radiation? Yes, military medicine in the atomic age will be radiological, but virtually hopeless and futile. There can be no medical service, no organized medical relief such as we prided ourselves in during World War II, for the medical service, the medical personnel, and the medical supplies for relief of misery are all vaporized in the holocaust.

Before the curtain of military security descended on the science of nuclear physics in America Dr. John H. Lawrence[2] delivered the Caldwell Lecture in this same auditorium in 1941 on the subject, "The New Nuclear Physics and Medicine," and showed the high promise held for many radioactive isotopes in cancer therapy and biochemical tracer techniques. His experiments had been carried out with radioactive atoms obtained from the cyclotron of his brother, Dr. E. O. Lawrence, at the University of California. Today the uranium pile is a far greater source of radioactive isotopes and this broad field for future research will now go forward much more rapidly. The lengthy symposium on this subject to be presented at the present meeting of our Society indicates the rapid strides already made since Dr. Lawrence's lecture. It is precisely this field where the teamwork of the physicist and those radiologists whose breadth of knowledge fits them for the task will offer most in the cooperative research work of the present and future.

However, it seems to me that a word of caution is in order against over-optimism concerning these new forms of radiation therapy. It is very easy for the lay public, and the medical profession as well, to be over-enthusiastic and over-optimistic at the present time because of the close association of radioactive isotope research with atomic energy development in the lay press and in the current popular science writings. The history of the therapeutic use of roentgen rays and radium is full of examples of over-optimism. Our distinguished colleague and fellow member of this Society, Dr. W. Edward Chamberlain,[3] expressed this idea exactly in a recent forum in which he participated. He said, "In the past we have been told too many times that this or that new and different (but not fundamentally different) source of radiation would revolutionize cancer therapy. There is no reason for believing, or hoping that these new methods of applying atomic energy therapeutically will provide a final answer to the cancer problem. . . . Cancer has been cured by irradiation in many thousands of cases and the percentage of such good results has increased steadily throughout the years. This improvement has been due in small part to improvement in equipment, in large part to increased knowledge and skill of the radiologist. These newer methods of releasing and applying atomic energy will doubtless prove of real value. The point is that we must not jump to the unwarranted conclusion that a revolutionary method has suddenly come to hand—one that will solve, or greatly modify, the problem of cancer." Many will recall the pre-war work on the therapeutic effects of direct neutron bombardment of cancer, and realize that much more clinical work is needed to fully evaluate neutron therapy. Neutron therapy and radioactive isotope therapy are gigantic problems yet to be solved after the interruptions of war.

Radiology in the Atomic Age will be greatly influenced by the vast post-war technological developments in another field of atomic research—electronics. The atom is also the home of the electron. Already the Morgan-Hodges photoelectric timer has

earned a valuable place in mass roentgenography and will soon find wide use in general roentgenography. Within the next few years many investigators will be studying the problem of amplifying the roentgenoscopic image several thousand times. Think of the unlimited possibilities of roentgenoscopy to which has been added a system of electronic amplification! With the illumination of the fluorescent screen approaching that of the roentgenographic viewbox in brightness, dark adaptation of the operator would be eliminated; examinations could be conducted in normally lighted rooms, the radiologist's office, the surgical amphitheater, etc.; the amplification would permit considerable reduction in the intensity of the roentgenoscopic exposure and markedly reduce radiation hazards. Also when the brightness levels in clinical roentgenoscopy become one hundred times their present value the stereoroentgenoscope will become practical and useful. The field of electron optics may make all this possible for radiology in the Atomic Age.

The construction and rapid development of the induction electron accelerator —the betatron—has opened whole new fields of study in the problems of supervoltage. At present there seems to be little or no limit to the energies which the electrons may attain in the Kerst betatron, and roentgen rays can be generated by its use which surpass anything known. Almost overnight the pre-war supervoltage range of 400 kilovolts and 1000 kilovolts with conventional roentgen tubes was exceeded, and papers are now appearing in our journals on the biological action of 20 million volt rays, doubtless soon to be followed by similar studies with 100 million volt roentgen rays. Will the betatron become part of the essential equipment of the radiologist in the Atomic Age?

Modern physical science, now a major cornerstone of radiology, has had a thousand almost unbelievable triumphs since the close of the last century. It has unlimited potentialities for helping man be man's best friend, and terrifying possibilities for helping man be his own worst enemy.

If the Atomic Age proves to be not one of mutual destruction of nations but a peaceful Age—and God grant that it will be—the frontiers of radiology will be advanced immeasurably. The precious time lost through the stupidities of war will be made up, and our specialty may well surpass the achievements of its first half century.

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SOME OBSERVATIONS CONCERNING EWING'S TUMOR SEEN IN AN ARMY GENERAL HOSPITAL*

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CHARACTERIZATION OF EWING'S TUMOR WITH ATTEMPT AT INTERPRETATION

AN EXCELLENT summary of the clinical and pathological manifestations of Ewing's sarcoma is that given by Kolodny[9] in 1927. This tumor is found most commonly in the small bones of the extremities, in the skull and shafts of long bones, differing in these respects from osteogenic sarcoma. In many instances the origin appears to be multiple, although this may be the result of the tendency of the tumor to become rapidly disseminated. As the tumor spreads through the haversian system, the cancellous bone is involved and the architecture disintegrates, so that tension and pressure lamellae are separated and compressed, and bone appears thicker in places. As the tumor approaches the cortex of the long bone, the latter becomes rarefied. In the more slowly growing tumors a secondary or tertiary shell of new periosteal bone is formed before the tumor perforates the periosteum, producing the appearance of laminae. More often the tumor advances too rapidly for periosteal reaction and undergoes necrosis and liquefaction resulting in a subperiosteal cavity filled with degenerated tissue resembling "pus." The consistency of the tumor depends on the stage of growth and presence or absence of regressive changes. At first it is soft, mushy, grayish white, and has the appearance of granulation tissue. This may easily mislead the surgeon into the belief that he is dealing with osteomyelitis. Later a soft, crumbly, marrowy growth is seen with connective tissue strands and frequent necrosis and liquefaction, producing the appearance of pus. Areas of yellowish cystic degeneration are found.

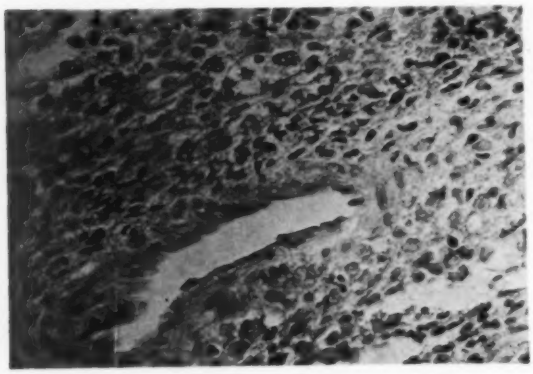

FIG. 1. Ewing's tumor, showing typical uniform small cell and its characteristic relation to blood vessels.

Histopathologically the tumor is characteristic (Fig. 1). The cell type is small, polyhedral and possesses little cytoplasm. The nucleus is round or oval. The appearance of pleomorphism is lacking and, even in the presence of abundant mitoses, tumor giant cells are not seen, in contrast to osteogenic sarcoma. The cells are extremely uniform and often form pavement sheets without organization. Not infrequently cells exhibit mucoid or hydropic degeneration. In many instances the cells are arranged around blood vessels, but this is not invariable and occurs also in other cellular sarcomas. Perhaps the most characteristic feature is the apparent complete absence of intercellular substance. As a result the cytoplasm of individual cells appears often to form a syncytium.

This tumor produces diffuse bone absorption and in its invasion of the surrounding soft parts causes corresponding shrinkage and atrophy of skeletal muscle fibers (Fig. 2). As a result the endomysium and

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perimysium become condensed into a trabeculated network acting as a framework for the growth of the tumor. In the presence of lymphocytic and plasma cell infiltration, the appearance is often very confusing and difficult to distinguish from that of a pure inflammation. The tumor spreads through the blood and lymph, especially the latter, and not infrequently involves the regional lymph nodes, but is most commonly characterized by involvement of all bones of the skeleton, especially the skull, again in contrast to osteogenic sarcoma.[12] The most frequent involvement

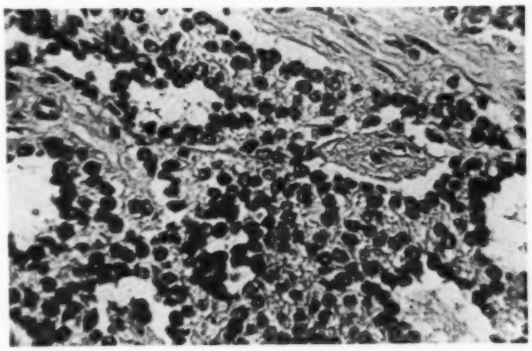

Fig. 2. Note tendency for the tumor cells of Ewing's sarcoma to creep along the perimysial framework of the overlying muscles bringing about atrophy of its fibers.

at autopsy is the lungs and skull. Metastases are histopathologically identical with the primary focus and behave in the same manner (Fig. 3). Ingrowth toward the dura may often produce pressure symptoms upon the brain, and epileptiform convulsions are not infrequent. Tumor cells may be demonstrated in the cerebrospinal fluid (Fig. 4).

Etiological factors include a decided sex predilection, about 3 cases out of 4 occurring in males, most often between the ages of five and fifteen. Occasionally the subjects manifest mild endocrinopathic disturbances, e.g. obesity or slim graceful bony structures

The location of the tumor is most common in the "pipe" bones, especially the tibia, fibula, humerus, ulna or femur in approximately that order. Occasionally

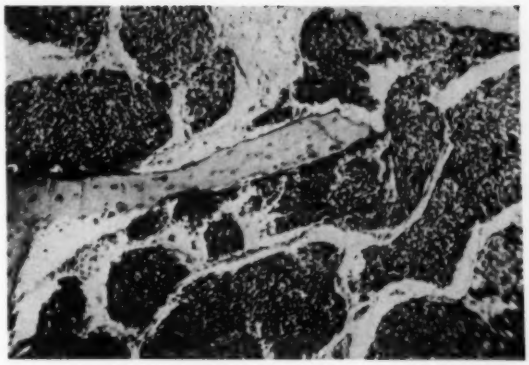

Fig. 3. Vertebral metastasis, showing solid sheets of tumor cells closely massed, with no apparent organization.

the clavicles, tarsal bones, ribs, vertebrae, mandible, skull, shoulder girdle and pelvis are involved. When a long bone is the seat of the disease, most or all of the shaft becomes involved. The history of trauma is not infrequent. Clinically the common first symptom is pain which is usually intermittent at first in contrast to osteogenic sarcoma. Frequently a long interval of freedom from pain may occur, lasting for several months. With each successive attack the pain becomes more severe and more prolonged and the interval shorter, until finally a permanent rheumatoid character of pain is established, usually with the appearance of a distinctly palpable tumor. Fever occurs with attacks of pain and occasionally is rather prominent, simulating the clinical picture of osteo-

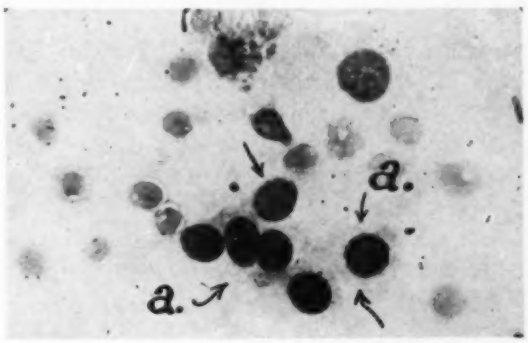

Fig. 4. Dural metastasis may be diagnosed by presence of Ewing's cells in the spinal fluid (postmortem lumbar puncture). Tumor cells at *a*.

myelitis or rheumatic fever. The tumor mass, when apparent, may be associated with considerable local hyperemia as well as enlarged regional lymph nodes. The tumor mass increases with each attack of pain until a permanent enlargement results. Joint involvement is rarely if ever seen. Pathological fracture is also rare until the terminal stages. The clinical course may be prolonged for years before the development of metastases; frequently, however, it may be only a few months' duration. With the development of metastases, especially within the lung, high fever may occur. Metastases are often first demonstrable in the skull. With or without radiation therapy remissions of many months may occur. Other viscera, such as the liver, kidney and spleen are involved, but rather infrequently. Among the clinical findings the following are encountered with surprising frequency: (1) history of trauma, (2) intermittence of symptoms including fever, (3) a mistaken diagnosis of chronic osteomyelitis in the case of long bones, or (4) of tuberculosis in the presence of the vertebral involvement.

According to Ewing[6] this tumor, which he classifies as "solitary diffuse endothelioma," causes widening and slow absorption of the shaft without a trace of bone production. The soft tissues are gradually invaded. The most striking feature, best demonstrated by the roentgen ray, is the involvement of a large segment or the whole of the shaft, which distinguishes the process from osteogenic sarcoma and the giant cell tumor. This location together with homogeneous demineralization of a slightly widened shaft, generally permits a diagnosis from the roentgenogram alone, especially if the tumor recedes rapidly under roentgen or radium treatment.

The histogenesis of Ewing's tumor is still subject to discussion among most authorities. Basically the tumor consists of solid cords of cells separated by fibrous bands, thick or thin. Delicate reticulin fibers may exist between individual tumor cells or may be absent. The cells generally are rounded unless closely packed, then are polygonal. The size is two to four times or more that of a small lymphocyte. The nuclei are rounded and the cell outline is indistinct. Often capillary blood vessels occur, either in septa or lying among the tumor cells. According to Oberling and others, reticulum cells are present; other authors deny this (Ewing and Foot). According to Stout[14] it is impossible to separate reticulum cell sarcoma and Ewing's tumor, and he agrees with Oberling that they are variants of the same tumor. Pseudorosettes may occur, which according to some may prove that these are neuroblastomas with bony metastases. This is denied by others. According to Oberling embryonal types of erythropoiesis and myelopoiesis may occur in some tumors and in his opinion the tumor is a reticulum cell sarcoma, most often seen as a cellular blastema without any differentiation or with differentiation into reticulin-forming tumor, endothelium-forming tumor, or a myelopoietic form, and so forth. This view is denied by Ewing. Stout, however, is inclined to Oberling's concept, particularly in view of the fact that Ewing himself admits that the process appears to begin in the perivascular endothelium. Neely and Rogers[11] agree with Melnick that the cell of origin is the undifferentiated mesenchymal cell which in postnatal life is found about small blood vessels and capillaries, typically being an undifferentiated round cell sarcoma, composed of cells not yet sufficiently mature to form reticulum. In atypical neoplasms in which the tumor cells are somewhat more differentiated, they may assume morphologic and physiologic properties of the fibroblast or fibrocyte, become spindle shaped, produce reticulum, and, with still more cellular differentiation, possibly form bone. Differentiation may also proceed along another line, producing reticuloendothelium and myelocytic elements and may resemble the microscopic picture found in lymphoblastoma. The cell of origin in their opinion, therefore, is the same as in fibrosarcoma, osteogenic sarcoma and

lymphoblastoma, and histopathologic classification depends on the line and extent of differentiation of the tumor cells.

Our own studies of cases at this hospital have provided little basis for formulation of an independent opinion. In some of the cases the relation of tumor cells to blood vessels is so characteristic that it is difficult to escape the conviction that the tumor is itself an endothelioma, as originally suggested by Ewing. Certain features of the histopathology of the disease deserve emphasis and may provide a basis for classification. These are (1) the lack of intercellular matrix, (2) the rudimentary cell type with slight if any differentiation toward a spindle cell, (3) the remarkable uniformity of the tumor cells both in the primary lesion and in the metastases, (4) the lack of marked anaplasia histopathologically despite the obvious malignancy of the disease, (5) the striking morphologic similarity between tumor cells and the cells of normal embryonal tissues in an exceedingly primitive stage of development, i.e., before the determination of cartilage, muscle, and other specific tissues from the undifferentiated mesoderm (Fig. 5). Because of the constant association of this tumor with a bone locale it seems necessary to relate this tumor in some manner to osteogenic mesenchyme. Many of the features listed above are characteristic of embryonal neoplasms in general. This is an important distinction for all tumors and should be kept in mind in any attempt at classification. As stated by Ewing,[7] "The embryonal nature of a tumor should not be confused with an anaplastic character. While many embryonal carcinomas are also highly anaplastic, others are relatively slow in growth and vary little from the cells of origin.The failure to distinguish between embryonal and anaplastic characters gives rise to much misapprehension regarding the prognosis of cellular carcinomas, since embryonal carcinomas are very prone to sudden variations in growth, they often respond to x-ray treatment, and their course is distinctly less malignant than that of anaplastic carcinoma of corresponding structure.

"Embryonal carcinomas must be conceived as arising from embryonal structures, often misplaced, and, since the cells of origin are imperfectly developed, the tumor reflects this character throughout. The embryonal character does not signify that there has been any reversion of the originating cells of the tumor from an adult to an embryonal type. It is possible to grade the carcinomas of many organs according to this principle, as adult or embryonal . . . ."

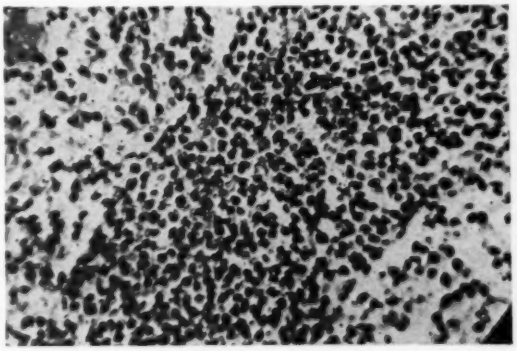

FIG. 5. This slide shows the morphology of cell structure in a 2 cm. fetus. Note the delicacy of structure and small size of the cells which are almost completely filled by their nuclei.

Inasmuch as an intercellular matrix is not apparent in this stage of development of the tumor cells one must anticipate the complete failure of the tumor cell, lacking a suitable substrate, to manifest any osteoformative properties. In any event it seems logical to interpret the disease, on circumstantial grounds at least, as a form of *embryonal osteoblastoma, nonosteogenic*, possibly bound up with certain dysontogenetic factors.

The value of such classification at this time is largely academic. Nevertheless, it may possibly open an avenue of approach to the eventual understanding of the disease in terms of constitutional diathesis and dysplasia (hamartia). Many parallels may be drawn between this tumor as an

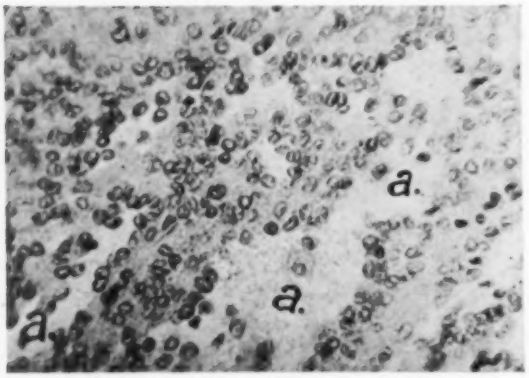

FIG. 6. Synovial sarcoma simulating Ewing's tumor. This was believed to be an embryonal synovioblastoma. Note clefts simulating rudimentary synovial spaces in a few areas, at *a*.

embryonal osteoblastoma and other embryonal tumors, for example embryonal carcinoma of the testis (seminoma) and neuroblastoma of the adrenal or kidney.[1] Examples may be multiplied to include many types of tumor, even certain tumors of synovia as in a case recently observed here in which the first impression was Ewing's tumor (Fig. 6 and 7). In all these tumors the characteristic feature is the monotonous regularity and uniformity of cell type, the rounded delicate nucleus, scanty cytoplasm, the syncytium-like formation of solid sheets of tumor in which the scanty cytoplasm is faintly stained or indistinct, and the lack of differentiation into any well defined structural units except perhaps occasional formation of rosettes and pseudorosettes, especially around blood vessels. In many of these cases, too, a dysontogenetic basis is often underlying.

ROENTGENOLOGIC AND THERAPEUTIC CONSIDERATIONS

In a recent survey of the literature concerning bone tumors it becomes apparent that there is considerable difference of opinion regarding the value of roentgen examination in diagnosis and as a guide in the choice of the method of treatment. We have reviewed the bone tumors which we have encountered since the opening of the Percy Jones General Hospital, January 15, 1943. To date there have been 30,442 admissions of which 55 were for such tumors, 24 of which were malignant, constituting a ratio of 1 malignant bone tumor to each 1,263 admissions. Comparison with the figures from a large civilian institution is of interest. Meyerding and Valls[10] state, "Bone tumors are rare in civilian practice, but comprise one out of every four thousand patients entering the Mayo clinic"; in other words, an admission rate of 0.025 per cent. In Meyerding's study of cases admitted between 1909 and 1934, 114 patients with Ewing's tumor were treated, i.e., approximately 27 per cent of 424 primary malignant tumors of bone encountered. According to statistics compiled by Swenson, 26 cases of Ewing's tumor were studied at Presbyterian Hospital between 1906 and 1942, a period of thirty-six years. This

FIG. 7. Cystic lesions and bone condensation changes in proximal third of the tibia. This was diagnosed from tissue sections as a synovioma simulating Ewing's tumor. The morphology was so embryonal that it was differentiated with difficulty from the classical picture of Ewing's tumor. Patient was a twenty year old T/5 who had pain in the right knee starting in September, 1943, and continuing in March and April, 1944, with recurring episodes of hydrarthrosis, following which a lump was noticed over the medial tibial condyle. Biopsy by Colonel Francis McKeever revealed bilobed cavity and an extraosseous mass which looked like neoplastic tissue. Bilobed cavity is shown at *a*. Histopathological findings are shown in Figure 6.

indicates that less than one case a year is seen in this large civilian institution. A survey of the cases in Percy Jones General Hospital shows that there is a very high proportion of Ewing's tumors, being approximately 42 per cent of malignant bone neoplasm encountered in this small series.

According to Rigler[13] there is great value in the roentgen examination of any suspected bone tumor. The roentgenogram ordinarily will indicate whether the tumor is intrinsic or extrinsic in relation to the shaft, whether it is benign or malignant and often will give an accurate index of the extent of involvement. In some cases the type of tumor present may accurately be determined. On the other hand, it has also been said "There is no such thing as a positive diagnosis of a malignant bone tumor with x-ray."

The above controversy is one in which we prefer not to become involved, for it is obvious that given a proper diagnostic examination and experienced interpreter, the roentgen findings are of inestimable value, and in some instances may actually be more diagnostic than the pathological report based on insufficient biopsy material (Fig. 20). In the triad of diagnostic features, the histopathologic, the bedside examination and the roentgenographic study, each plays a necessary part and no one of these should be neglected. Early, accurate diagnosis depends upon the proper interpretation of all three factors which we believe are complementary and not antagonistic. As is stated by Brailsford,[2] it seems reasonable to assume that " . . . a positive finding in any one should be regarded as a dominant factor."

One source of difficulty in reaching an early diagnosis in Ewing's tumor is the fact that very often these patients have rheumatic pains and various ill defined complaints and are classified as "goldbricks" by the uninitiated and given liniment, hot packs, and sometimes the well known "brush-off." It would seem axiomatic, therefore, to state that a meticulous history and physical examination are imperative in individuals with vague complaints, and emphasis should be placed on repeated roentgenographic examinations whenever there is doubt regarding diagnosis.

The following case histories stress the importance of an early roentgenographic examination:

Case I. A white male private, aged twenty-one, was admitted to Percy Jones General Hospital, December 23, 1943. He had been inducted into the army December 5, 1942, but prior to this time for five or six years had complained of intermittent shooting pains in the right thigh. Pain was most severe in the region of the knee and the patient complained of a dull gnawing ache in the mid-thigh. Three days following induction he reported to sick call and after numerous visits was finally hospitalized May 8, 1943. He was examined at a Station Hospital for foci of infection, but no roentgenograms were made. In September, 1943, nine months after induction while on maneuvers he turned to get a shovel from a truck and suffered a complete pathological fracture of the mid-portion of the right femur. Roentgenograms made at Brooke General Hospital in Texas revealed a tumor and biopsy showed Ewing's sarcoma. He was transferred to this hospital for deep roentgen therapy. The patient received treatment to multiple fields, but after a retrogressive course ending in debilitation the patient died September 26, 1944, nine months after admission (Fig. 8, 9 and 10).

Case II. A white private first class, aged twenty, was admitted to Percy Jones General Hospital August 27, 1943, with a complaint of pain in the right heel, first noted in March, 1943. At this time he reported to his dispensary, was told that he had a sprain and to "forget it." A week later, the pain persisting, the ankle was strapped, but no improvement was noted. In April, 1943, he entered a Station Hospital for repair of a hernia. His foot continued to pain. In May he was transferred to another Post where, following strenuous exercise, he again found it necessary to report to the dispensary. He was thereupon hospitalized and treated with "hot packs" for eighteen days, following which biopsy was done. This revealed a Ewing's tumor. The patient was then transferred to this hospital where heavy preoperative roentgen therapy to the os calcis and 100 r (air) to the

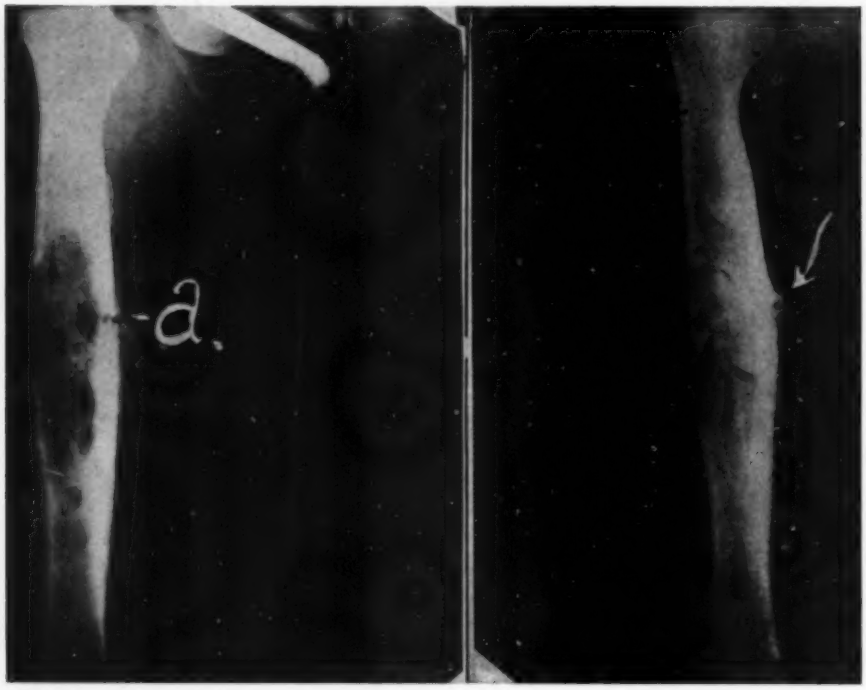

FIG. 8. Case 1. Ewing's tumor of femur involving 32 cm. of the shaft. Note expansile character of the lesion and multiple lucid areas involving the distal two-thirds of the shaft. A pathological fracture is seen at *a*. On the right the appearance following 2,000 r to each of four fields covering the entire shaft of the femur. Note the marked improvement in appearance following deep roentgen therapy.

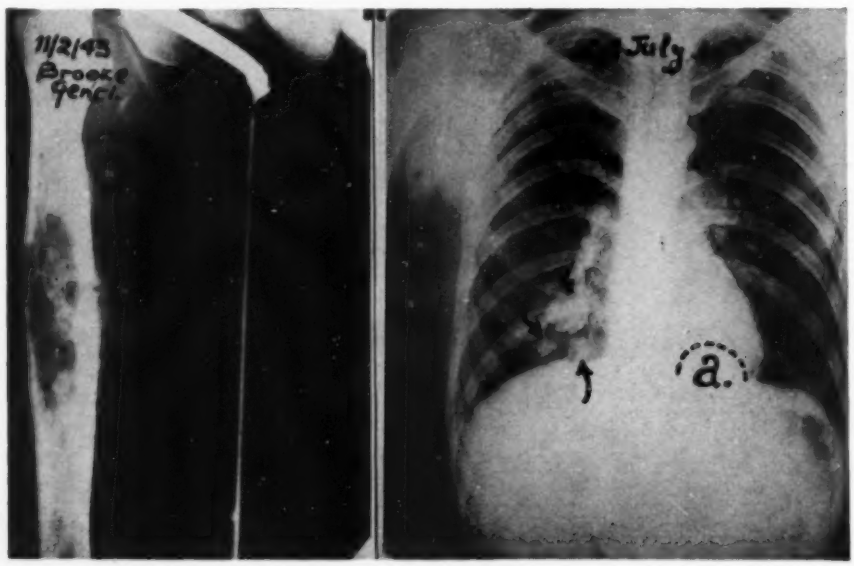

FIG. 9. Case 1. Shows the femoral shaft in original films made at Brooke General Hospital on 11/2/43. On the right the appearance of the chest seven months later. Numerous metastatic nodules are evident in the right lung field. There is a large metastatic lesion behind the cardiac silhouette at *a*. Close inspection of the cardiac silhouette and the domes of the diaphragm is imperative. Not infrequently the earliest metastatic deposits will be found in the costophrenic sulci.

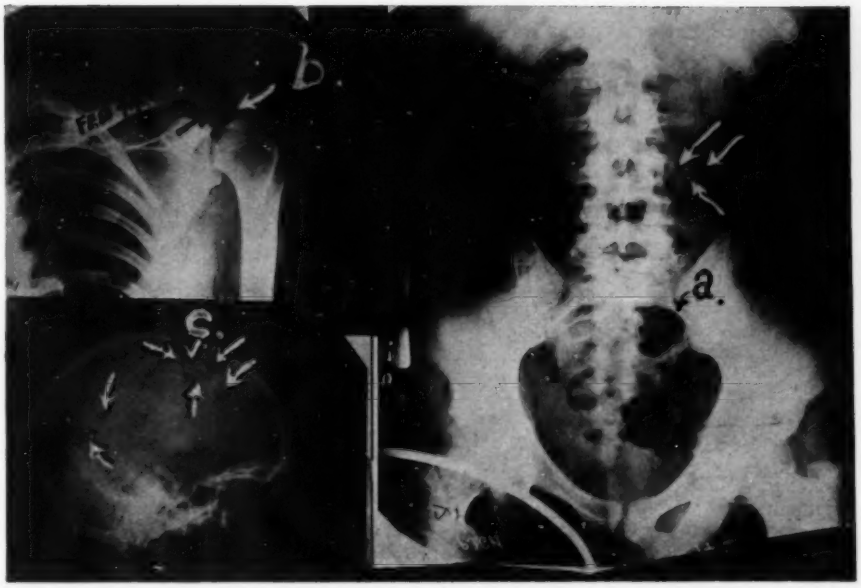

FIG. 10. Case 1. Ewing's tumor, same case as Figures 8 and 9. This shows metastatic lesion in left acromion. Film made in February, 1944; lytic lesion is indicated at *b*. In May, 1944, a large defect was found in the sacrum at *a* and the left transverse process of the fourth lumbar vertebral body was subsequently found to be completely destroyed, at arrows. At the same time metastatic disease became evident in the calvarium as shown in the left lower illustration. Lesion at *c* looked very much like a venous lake, but a nodule could be palpated in this region on clinical examination.

entire thorax was given, following which on October 28, 1943, a Gritti-Stokes amputation was done. No viable tumor cells were found in the resected specimen (Fig. 11). The patient was discharged to a Veteran's Facility, but in July, 1944, nineteen months after the beginning of his illness, metastatic nodules were found in the left lung field. He died at home of "cardiac failure" November 7, 1944 (Fig. 12, 13, and 14).

Pitfalls in Roentgen Diagnosis. Much to the credit of forward echelons, very early diagnoses of bone sarcomas have frequently been made and substantiated by subsequent biopsy. On the other hand, obvious errors are also apparent. In one instance a lytic lesion of the femoral condyle was interpreted as an "eosinophilic granuloma" and in another the "sun-ray" periosteal reaction of an invasive, rapidly growing osteogenic tumor was construed to indicate the presence of callus about a fracture which was nonexistent (Fig. 15). As pointed out by Sutherland[15] failures in arriving at a proper diagnosis " . . . must be attributed to a lack of knowledge and experience on the part of some diagnosticians and carelessness in the use of knowledge and experience on the part of others." Such failures should not be blamed on the roentgen method.

It is quite certain that early in the growth of Ewing's tumor the roentgen findings are not characteristic enough to warrant a positive diagnosis, but obscure lytic changes in bone should be followed by serial roentgenography and early biopsy. Minimal periosteal proliferations should be looked upon with concern and not considered to represent "osteomyelitis" until proved otherwise, for it is well known that roentgenographically and clinically, Ewing's endothelioma closely simulates nonspecific bone infection from which it is differentiated with difficulty.

One type of Ewing's tumor likely to give trouble in roentgen diagnosis is that located in the sacrum. In the differential diagnosis of sacral tumors, among other lesions one must consider chordomas, located centrally, neurofibromas extending along the course

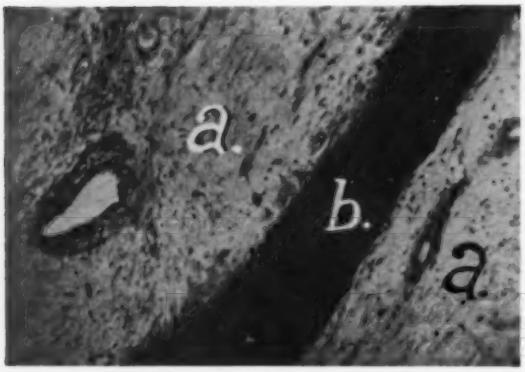

FIG. 11. Status after irradiation, showing disappearance of tumor cells. The remaining cells resemble embryonal mesoderm as seen in Wharton's jelly. Residual myxomatous tissue at *a*. Bone spicule at *b*.

of the sacral nerves and ependymal cell gliomas. According to Camp and Good, Ewing's tumor is far from characteristic in the sacrum being much more typical when involving long bones. With Ewing's endothelioma of this portion of the pelvic girdle, the roentgenologist can only make the diagnosis of malignant disease. The following case report emphasizes the difficulties in diagnosing a Ewing's tumor of the sacrum.

FIG. 12. Case II. Ewing's tumor in twenty year old Pfc. with history of painful heel of five months' duration, showing Ewing's tumor involving the posterior portion of the os calcis. There is no sharp line of delineation between the tumor mass and the surrounding normal bone. Tumor at *a*.

CASE III. A thirty-nine year old T/5 was referred to the Roentgen Therapy Section of

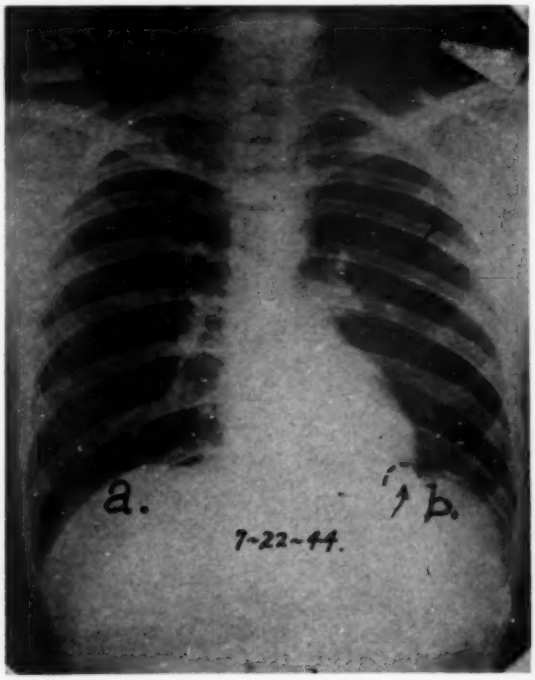

FIG. 13. Case II. Showing early metastatic lesions in left lung field. Careful inspection of the regions indicated at *a* and *b* is most important in order to exclude early metastatic sarcoma behind the domes of the diaphragm. In this particular case a faint metastatic deposit was noted behind the apex of the heart. The nodule is indicated by the lower arrow.

Percy Jones General Hospital for treatment on November 13, 1944. He gave a history of severe lancinating pains in the right buttock of seventeen months' duration. Four months before there had been a sudden increase in the pain after the patient was thrown from his feet. Roentgen study revealed a large tumor arising from the right hemisacrum in the region of the anterior sacral foramina, some of which appeared dilated. On the basis of previous experience with neurocutaneous syndromes and von Recklinghausen's disease the diagnosis of

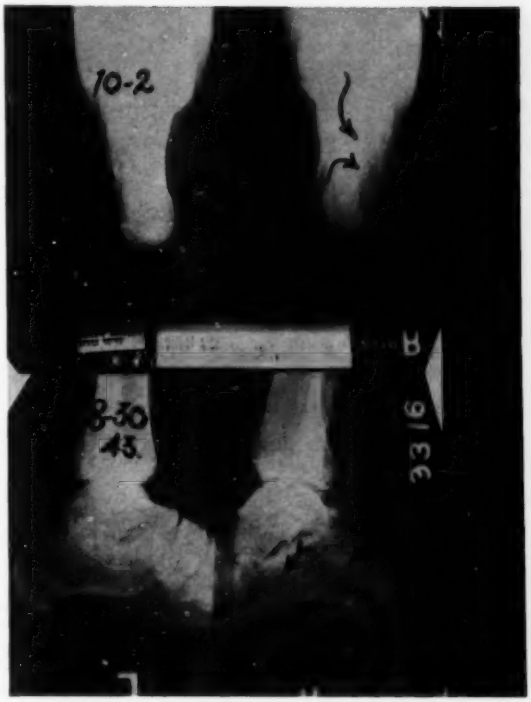

Fig. 14. Case 11. Showing appearance of the right os calcis in the axial view with left side for comparison, above. Lateral projections, below. When this patient reached us there was a large soft parts mass which had burst through the shell of the os calcis. Lytic areas in the right os calcis are indicated by arrows. Histopathological findings are shown in Figure 1.

neurogenic sarcoma was suggested. We felt that a neurofibroma or an ependymal cell glioma were possibilities and that a chordoma was not very likely. A punch biopsy was attempted, but was unsuccessful (Fig. 16).

The patient was given a small amount of palliative roentgen therapy (800 r to a large sacral field) and referred to the Orthopedic Section for biopsy, and possible surgical removal. At operation, November 22, 1944, the neoplasm was found to be inoperable, but several specimens were obtained and the following report rendered by one of us (S.E.M.): "It appears we are dealing with a sarcoma possessing embryonal characters, i.e. round nuclei, scanty delicate cytoplasm and syncytial arrangement in compact masses. Anaplasia is relatively slight and mitoses are few. There is abundant necrosis and hyalin scarring. It is difficult to assign a derivation to the tumor, but from the morphology of the cells, including their embryonal aspects, their lack of anaplasia and the marked abundance of thick-walled, poorly formed blood vessels, the most plausible

Fig. 15. Characteristic roentgen picture of osteoblastic-osteogenic sarcoma of the proximal third of the right humerus. Note Codman's triangle at *a* and bone production in the axillary lymph nodes at *b*. This profuse type of "sun-ray" proliferation should not be mistaken for abundant callus. The fact that the lymph nodes show evidence of bone production in this region proves that the primary tumor cannot be Ewing's sarcoma since Ewing's tumor is nonosteogenic.

FIG. 16. Case III. Ewing's tumor of sacrum. Soft tissue tumor at *a*. This large mass displaced the rectum anteriorly, and appeared to rise from the region of the anterior sacral foramina. The roentgen picture is not characteristic. The following diagnoses were considered: neurogenic sarcoma, neurofibroma, ependymal cell glioma, chordoma. The lateral position of the tumor would tend to rule out the last diagnosis.

diagnosis is Ewing's tumor." Additional deep roentgen therapy was given between December 4, 1944, and March 13, 1945, to each of four large fields receiving 3,000 r and a lateral sacral field received 800 r (air). Patient is asymptomatic at present time (two years and one month since onset of symptoms).

The above case requires some comment. This lytic lesion of the right sacrum was considered on the basis of the roentgenogram to be some type of malignant tumor. The appearance was distinctly unusual for Ewing's tumor, the lesion apparently being sharply circumscribed. In this case we were unable to determine roentgenographically the type of tumor present, although review of some of our material indicates roentgen diagnosis can be highly accurate. It is enough, in our opinion, to call attention to the fact that a tumor is probably malignant without identifying the type of cell present; on the other hand, the clinicoroentgenological findings are often such that the type of osteogenic sarcoma can be surmised. This, of course, does not mean that we are making a "positive diagnosis" of the cell type, but are merely calling attention to the possibilities and probabilities on the basis of appearance and previous experience. If there is any probability of a lesion being Ewing's tumor, the importance of the therapeutic test should not be overlooked. Small doses of 100 to 150 r will often cause rapid regression of the tumor so that after as few as two to three treatments the patient's night pain is completely relieved. According to Cutler, Buschke and Cantril[5] the therapeutic test " . . . is often superior to histological examination in its diagnostic significance." The radiosensitivity of Ewing's tumor places it apart from all other bone tumors.

In regard to the other osteogenic tumors, it has been our experience that relief of pain either does not take place with a reasonable amount of roentgen therapy (1,500 to 2,000 r to multiple fields) or else the analgesic effect is delayed for a matter of two to four weeks. If the lesion involves a considerable extent of the shaft of a long bone with slight evidence of periosteal reaction to begin with, but following therapy shows evidence of considerable deposition of calcium in the periosteal and subperiosteal region and if at the same time the patient has had marked relief of pain within two to four days after the onset of deep roentgen therapy, the diagnosis of Ewing's tumor can usually be made without hesitation. The following case report is an example of the response of a Ewing's sarcoma to roentgen therapy:

CASE IV. A white officer, aged twenty-eight, was admitted to the Percy Jones General Hospital on October 25, 1944, with a complaint of swelling of the left leg and muscle fatigue of approximately one year's duration. The pain was not severe and was attributed to strenuous marching. One month prior to admission he noticed tumefaction of the leg, but the pain remained at a minimum. He reported to the dispensary and was told to apply "hot packs."

When first seen in the Roentgen Department he was complaining of severe pain in the left leg. Roentgen examination made just prior to this time showed a diffuse increase in the diameter of the fibula extending from a point about 2 inches distal to the proximal fibular head downward to the junction of the middle and distal thirds. The following comment was a part of the reports of the roentgen examination: "The bone in this area is definitely irregular in density. There is a calcification beneath the periosteum and there is a definite suggestion of destruction of the cortex in the anterior one-third of the fibula. These changes strongly suggest the presence of a malignant tumor. We believe that a so-called Ewing's tumor is more likely than an osteosarcoma. The other bony structures seem to be normal" (Fig. 2 and 17).

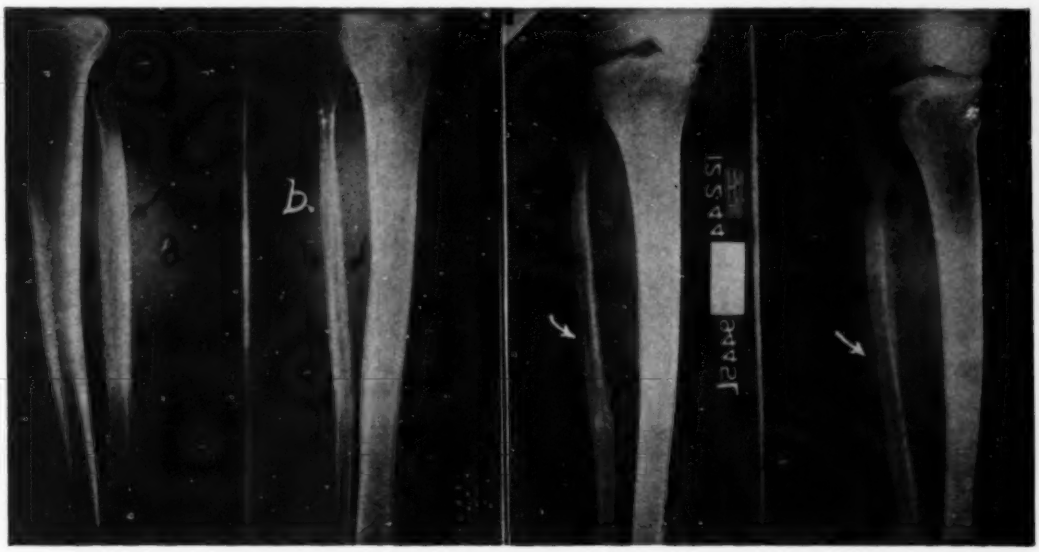

Fig. 17. Case IV. Ewing's tumor of fibula. Soft tissue tumor at *a*. The compact bone of the middle third of the fibula is infiltrated and honeycombed by tumor cells. There is a slight constriction of the fibula at *b*. Very slight periosteal reaction is noted in the extreme right film just adjacent to the arrow. This patient, a twenty-five year old Captain in Ordnance, gave a history of "muscle fatigue" of one year. Severe pain did not play an important rôle in this patient's history. Marked improvement followed roentgen therapy which was followed by mid-thigh amputation.

Roentgen therapy was completed on November 28, at which time the patient had received 2,000 r (air) to each of two fields crossfiring the proximal two-thirds of the left leg. On December 4 re-examination of the left lower leg showed quite extensive calcification along the margins of the involved portion of the fibula and a marked decrease in the size of the mass in the soft parts. The patient had been completely free from pain since his second treatment. On December 5, 1944, an amputation of the left leg was carried out by the Orthopedic Department. At this operation a tourniquet was placed around the leg at the level of the popliteal space and another tourniquet around the leg at the highest possible level of the thigh. An incision made over the fibula revealed a tumor mass which grossly showed evidence of invasion of the muscles and was obviously malignant. Without removing either tourniquet an amputation was carried out through the middle third of the femur, cutting equal anterior and posterior flaps. The patient's postoperative course was uneventful and no metastases have been found to date.

Pathological diagnosis: Ewing's tumor, left fibula, invading muscle.

Comment. An exact diagnosis was made on the basis of (1) the response to roentgen therapy, (2) a rather characteristic roentgen picture with evidence of deposition of calcium in the tumor after adequate therapy. Amputation was done without biopsy, but only after ascertaining that there was evidence of malignant disease invading muscle.

We feel that in a *doubtful case* biopsy should always be employed; however, it is worth while to recall that Ewing[7] has stated in regard to osteogenic sarcomas in general that treatment " . . . is a difficult and complex subject," and also, that if the simple plan of biopsy and amputation is carried out indiscriminately " . . . it will soon appear that many unncessary and unsuccessful operations are performed, several important aids in diagnosis, prognosis and treatment now available will be ignored and the best interests of the patient are not conserved."

Another example of the efficacy of roentgen therapy in establishing a diagnosis of Ewing's tumor is as follows:

CASE V. On March 22, 1944, a First Lieutenant aged twenty-three, was referred to the Roentgen Therapy Department of our hospital for consultation. He gave a history of pain starting in the low back and joints of the right leg while moving cases of beer for his father in 1940. This pain was "toothache like" in character. It was more pronounced in the joints. In June, 1942, after finishing primary training, he noticed pain in the back and legs which caused him to limp. He was given some pills and light therapy at this time and the pain disappeared. In August, 1943, his teeth were checked and no foci of infection were found. In January, 1944, he noticed a slight swelling of the right thigh while in the shower, and was immediately transferred to Letterman General Hospital where roentgen therapy was given to multiple fields (six) crossfiring the right thigh, 1800 r (air) being given to each of six fields measuring 10×15 cm. in diameter. The pain in the back and joints of the leg left forty-eight hours after the beginning of treatment and this swelling subsequently disappeared.

Roentgenograms showed a widening of the shaft in the proximal half of the right femur with "onion-peel" and "sun-burst" periosteal reaction indicating some form of irritation of the subperiosteal space. It was our opinion that if bone infection had been present and 1,800 r had been given to each of six fields a severe reaction would have resulted. Instead his symptoms promptly subsided. In view of the fact that there was a definite skin reaction outlining the various fields used, it was decided that no additional treatment was indicated and that biopsy should be considered.

Diagnosis of the Roentgen Therapy Department: "Ewing's tumor." Colonel Francis McKeever concurred in this opinion and it was decided by the Tumor Board to do a biopsy which was carried out on March 29, 1944. A large block of bone was removed from the lateral aspect of the femur at the site of the lesion. This measured 1×3 cm. and included periosteum, subperiosteal tissue, cortex and medulla. The specimen was seen by the pathologist who deemed it adequate and a report dated April 25 was as follows: "Dense scar tissue and fragments of bone undergoing absorption and necrosis. No evidence of tumor seen" (Fig. 18).

The patient was discharged from the hospital with the following diagnosis: "Osteomyelitis, chronic nonsuppurative, femur—right, upper third. Moderate. Cause—undetermined."

It was felt that he should be restricted to the continental limits for duty. The patient was traced by letter and on March 14, 1945, the following communication was received from the Chief of the Orthopedic Section at Letterman General Hospital:

"As you remember, Lt. —— was returned to duty by the Disposition Board and sent to Santa Ana Air Base. Seven days after his return, while playing baseball he sustained an oblique fracture of the right femur through the pathological area in the region of the biopsy. He was hospitalized. From that time until the 1st of November, 1944, when he was readmitted to Letterman, his fracture was treated by skeletal traction and healing of the fracture was quite slow. In September, 1944, he developed symptoms and signs compatible with a cerebellopontine angle tumor. An exploration at the Station Hospital revealed an invasive neoplasm which was diagnosed by the Army Medical Museum as 'probable myeloma, radiosensitive.' Chest x-rays now revealed pulmonary metastases and a course of palliative x-ray therapy was instituted. At postmortem no tumor cells were found in the femur and the x-ray department considered the primary cured, but death from pre-existing metastasis occurred."

This case report stresses again the importance of the therapeutic test in Ewing's tumor and shows that even in spite of a negative biopsy that the original lesion was an endothelial myeloma which later metastasized to the chest and brain.

There are a number of other pitfalls in diagnosis which should be mentioned,

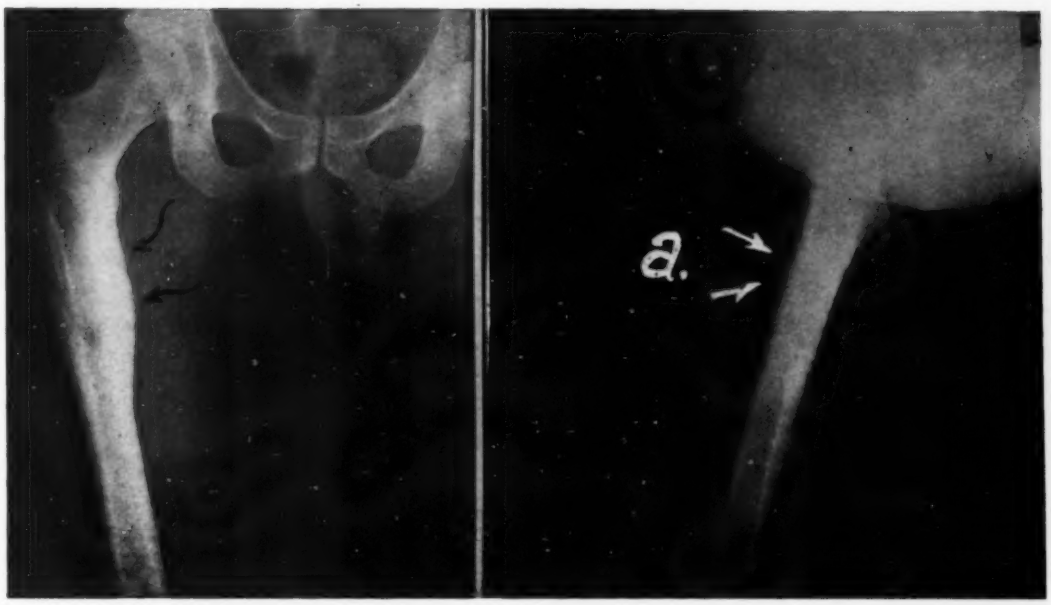

Fig. 18. Case v. Ewing's tumor in twenty-three year old Air Force Lieutenant with history of toothache-like pain in the right leg since 1940. Treated at Letterman General Hospital with deep roentgen therapy with prompt relief of pain and swelling of right thigh. Because of the dramatic response to treatment a diagnosis of Ewing's tumor was made. Subsequently a very adequate biopsy was obtained. Pathological diagnosis: chronic nonsuppurative osteomyelitis of the right femur. The patient returned to duty, but later developed signs of increased intracranial pressure due to a possible cerebellopontine angle tumor. Death occurred November 6, 1944. Postmortem revealed evidence of metastases to brain, skull, lungs, pleura and dorsolumbar spine. No tumor cells were found in the primary lesion of the femur, which had been completely controlled by 1,800 r to each of six fields cross-firing the lesion. Minimal periosteal reaction was observed at *a* on original roentgenograms.

among which are intercurrent disease such as syphilis. In one of our cases a history of chancre together with roentgen evidence of spontaneous regression in the size of the tumor made diagnosis difficult. It is well to remember that in Ewing's tumor there may be spontaneous increase and decrease in the size of the lesion. This may explain the well known clinical observation of alternating episodes of pain interspersed with comfortable periods. The following case history is illustrative:

Case vi. A private, aged twenty-four, was admitted February, 1944, stating that three years before admission he struck his left foot in an army truck accident. He strapped his foot and continued on duty. Six months later considerable pain and swelling were noted in this area and this became progressively more prominent. In October, 1943, a biopsy was obtained and a pathological report of hemangioendothelial sarcoma was made and confirmed by the Army Medical Museum.

Shortly after admission to this hospital it was noted that the tumor had regressed and that the bone lesion had apparently healed. It was then discovered that the patient had a history of chancre in July, 1942, and in view of the fact that a luetic lesion may mimic any type of bone lesion, it was felt advisable to repeat the biopsy before treatment was instituted. This was done and the original diagnosis was confirmed. The patient was given roentgen therapy between March 17 and May 2, 1,800 r (air) being administered to two large fields, cross-firing the great metatarsal and surrounding soft tissue region. Following this treatment the patient complained of moderately severe pain in the foot extending up the left leg. On May 5 an amputation was performed, through the middle third of the left lower leg and a prosthesis was fitted July 8, 1944. No viable tumor cells were found in the resected specimen.

The patient was discharged. He is living and is well at this time with no evidence of pulmonary metastasis, but complains of occipital headache. So far no metastases have been demonstrated in the cranium (Fig. 19).

It should not be inferred from the above cases that all Ewing's tumors are radiosensitive. Neoplasms vary in sensitivity according to inherent characteristics which often are little understood. Although most of the embryonal round cell sarcomas which have been treated in our clinic have been at least temporarily radioresponsive, experience in roentgen therapy teaches that there are exceptions and that even very embryonal tumors are sometimes radioresistant. The reverse is also possible; i.e., a very well differentiated tumor may respond dramatically to deep roentgen therapy as it did in the following case.* The reason for this is not clear, but probably depends upon certain factors having to do with the vasculoconnective tissues of the tumor bed and the patient's general resistance. Indeed, the only true measure of radiosensitivity is a test by thorough irradiation.

CASE VII. A patient, aged twenty-three, was admitted to Percy Jones General Hospital June 14, 1943, with a history of pain in the left leg of two years' duration. Tumor of the femur was discovered six months ago at Letterman General Hospital. The patient had lost 40 pounds in weight and the muscles of the left thigh were considerably atrophied. Roentgen examination showed an irregular punched-out lesion of the middle third of the femur surrounded by a fusiform expansion of cortical bone. Original roentgenograms suggested primary bone tumor or sclerosing osteomyelitis. Biopsy at Letterman General Hospital reported " . . . atypical telangiectatic osteogenic sarcoma or malignant angio-endothelioma of the femur." However, on the basis of tissue submitted to the Army Medical Museum for review it was felt that amputation was not indicated. A benign lesion was suggested, possibly an "ossifying angioma of bone." The original sections were reviewed in our Pathology Department and it was felt that in view of the night pain and sclerotic changes plus the

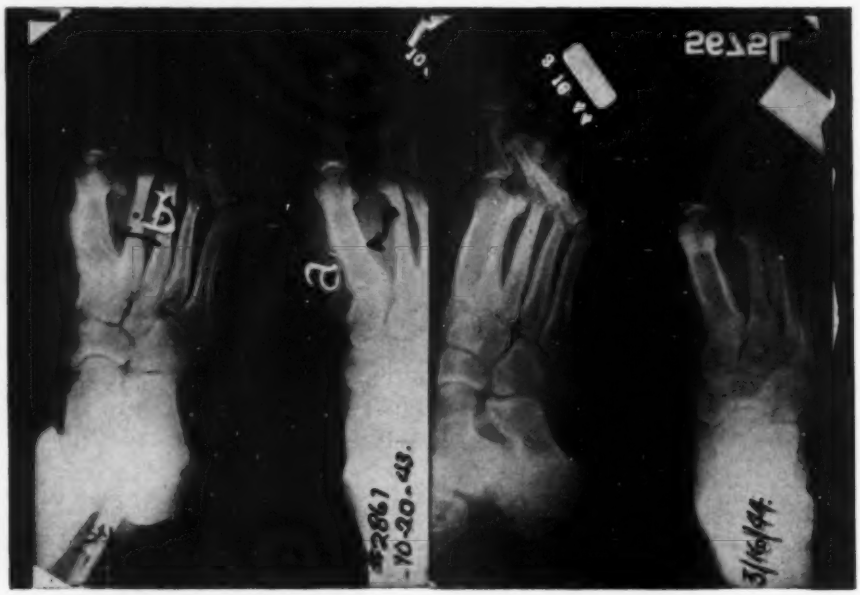

FIG. 19. Case VI. Ewing's tumor involving left great metatarsal. A pathological fracture is seen at *b*. The shaft is increased in width and there is a soft tissue tumor at *a*. On the right the appearance of the foot following surgical biopsy done elsewhere. The soft tissue tumor has regressed and the great metatarsal fracture has healed. Spontaneous regression of Ewing's tumor will sometimes occur, leading one to the erroneous impression of a benign lesion.

* Previously reported by Bell, J. C., and Heublein, G. W., in *Radiology*, 1944, *43*, 425–485.

appearance of atypical osteoid with patchy calcification that this might represent an "osteoid-osteoma." However, after reviewing all films the Roentgen Department on June 16, 1943, rendered the following report: "Considering all the films in this case it would seem that we are dealing with a primary malignant bone tumor, probably an osteogenic sarcoma of some type." The tissue sections were reviewed by two eminent pathologists, one of whom considered the sections quite typical of osteoid-osteoma while the other suggested the definite possibility of a metastatic lesion, having origin in the kidney. Numerous intravenous urograms were done and no evidence of a renal tumor could be demonstrated.

On July 30, 1943, the lesion was again examined and it was felt that we were dealing in all probability with a primary bone sarcoma. The patient's pain recurred and he was referred to the Roentgen Therapy Department for treatment which was administered between January 5 and 27, 1944, 1,800 r (air) being delivered to each of two large 15×20 cm. fields covering the lesion. Three days after treatment was started it was noted that the patient still complained of pain, but on the twelfth day of therapy he stated he was definitely more comfortable. Amputation was done February 24, 1944. The specimen showed an infiltrating malignant tumor which was finally considered to be characteristic of metastatic adenocarcinoma, the primary site of which could not be determined. The patient was carefully followed, but even after review we were unable to show any evidence of a primary tumor in any other organ.

Following amputation the patient gained considerable weight and his general condition was better than any time since admission to the hospital. It was felt that he had received maximum benefit and he was given a Certificate of Disability Discharge, with a final diagnosis of carcinoma, scirrhous adeno, middle third—left femur. The final laboratory pathological report was as follows: "Metastatic scirrhous adenocarcinoma with unusually abundant osteoid stroma and dense involucrum formation" (Fig. 20).

It was felt the most likely possibility of the primary focus in the order of probability was (1) renal tumor, (2) body of pancreas, (3) stomach, (4) bronchus or (5) prostate.

Comment. This case is of interest because it shows the difficulties that are sometimes encountered as the result of insufficient biopsy material and the importance of the careful roentgen investigation which suggested the presence of malignant disease of bone.

Fig. 20. Case vii. Tumor of the left femur, diagnosed as osteoid-osteoma on the basis of an incomplete histopathological section. This case responded to roentgen therapy after approximately twelve days' treatment. Roentgen diagnosis: malignant tumor of bone. Following amputation this proved to be "metastatic scirrhous adenocarcinoma" with unusually abundant osteoid. No primary source of the tumor could be found after careful investigation. Exposure determined by means of the Morgan meter.

It will be noted from the case reports presented, that roentgen therapy has been used as an adjuvant to orthopedic treatment in this hospital. We feel that bone sarcoma is primarily a surgical disease, and treatment by deep roentgen therapy alone is not warranted except in those cases where the lesion is inoperable or where the

patient refuses surgical treatment. It is difficult to escape the conclusions of Brunschwig and Tschetter[3] that " . . . no tumor is surgically resistant, but there are many tumors that are radioresistant and radical removal should always be urged upon the patient first."

Certainly roentgen therapy must be individualized, but whether it is necessary to attain a maximum tissue dose of 4,500 r (air) as suggested by Swenson[16] in his excellent article is problematical. Woodard and Higinbotham[17] have pointed out that the phosphatase producing mechanism of most osteogenic tumors is inactivated by radiation therapy when the tissue dose is in the region of 4,000 r. One may be justly excused for speculating why osteogenic tumors considered highly radioresistant, except in children, should be inactivated at 4,000 r plus and why the relatively radiosensitive tumors such as Ewing's tumor should require a similar dosage, 4,500 r. Brunschwig and Tschetter have indicated that as high as 30 per cent of bone tumors can be inactivated (not cured) by dosages ranging between 2,400 r (air) delivered in six weeks to 13,000 r (air) over an interval of five and a half years.

The major problem, particularly in Ewing's tumor, is to prevent the appearance of chest metastases which usually make themselves manifest within two years of the time treatment is instituted. Woodard and Kenney[18] have suggested the use of radioactive phosphorus as a method of inhibiting the implantation and growth of tumor emboli. It is possible that this tret-a ment may play a very important rôle in the control of metastatic disease at the time when P^{32} again becomes available. It is possible also that some form of total body irradiation as advocated by Heublein[8] or general therapy over the thorax carried out at long distance with low intensity over a period of days or weeks may control the appearance of metastases and improve the five year survival rate.

COMMENT

Despite its mimicries by other types of small round cell malignant growths, Ewing's tumor remains a well defined clinical and pathological entity. Intermittent fever, bone pain, and localized swelling are its earlier clinical manifestations, frequently preceded by an incident of trauma. With the eventual development of a definite tumor mass the roentgenological appearance is distinctive and often permits diagnosis, particularly when confirmed by rapid subsidence of the tumor following radiation therapy. Histopathologically the tumor is also well defined and can ordinarily be diagnosed with reasonable assurance by any experienced pathologist. The cell type is distinguished by its lack of variation from its basic pattern even in its metastases, by the uniform clustering of cells into solid sheets without appreciable architecture except that provided by blood vessels or the framework of invaded tissues, by the paucity of mitoses or other "malignant" features seen in more anaplastic tumors, and by the delicacy of structure of the cell body and nucleus. All of these features suggest an embryonal type of tumor and point to the usefulness of employing this category of classification of tumors not only for Ewing's tumor, but for similar undifferentiated malignant growths which may imitate it, such as neuroblastoma of the adrenal medulla, seminoma of the testis, and certain others. The embryonal nature of the tumor and its predilection for the earlier age groups possibly reflect an underlying element of dysontogenesis, i.e. faulty development, in its etiology in accordance with the "embryonal rest" theory of tumor origin.

SUMMARY

1. Patients with bone tumors constitute a relatively high percentage of admissions to Percy Jones General Hospital. This is in part due to the age group with which we are dealing and in part to the fact that this is an Amputation and Deep Roentgen Therapy Center.

2. Roentgenographic examination is of inestimable value in the diagnosis of all types of bone tumors, including Ewing's

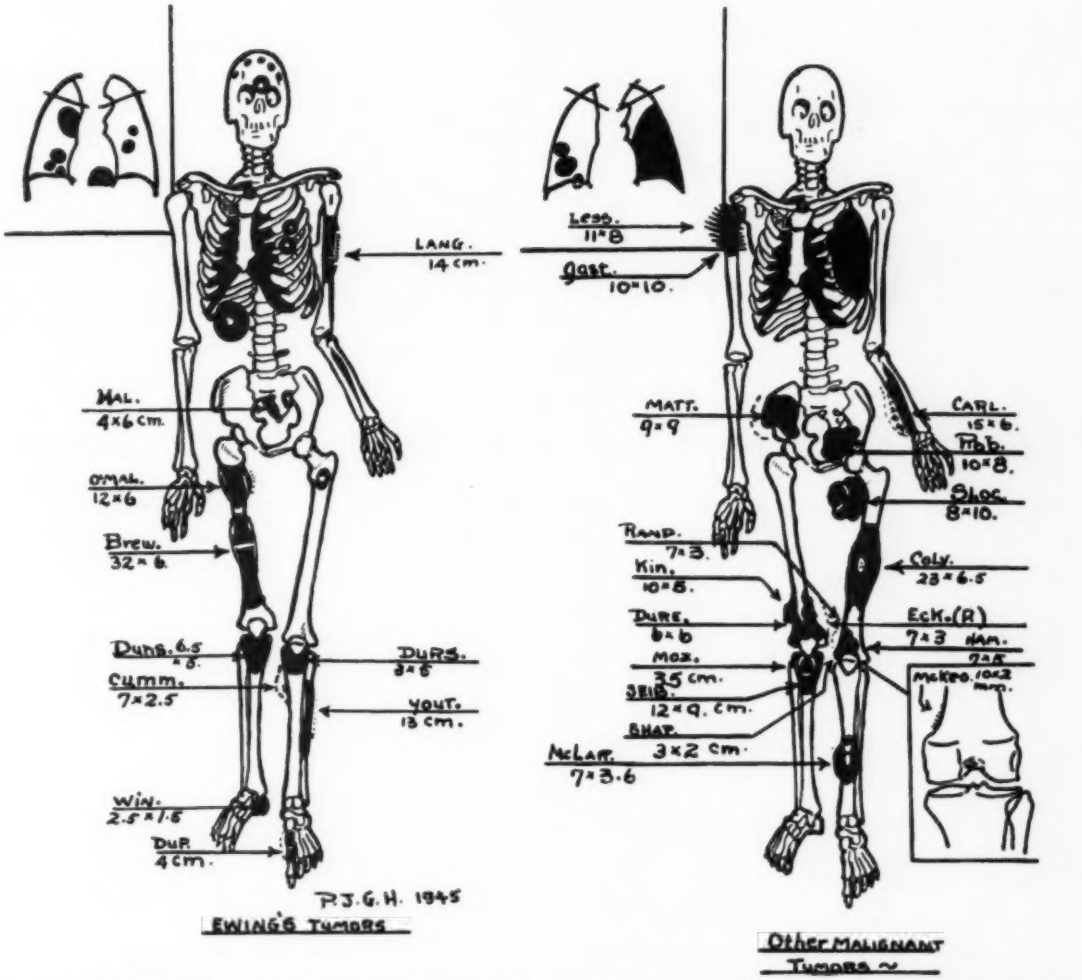

FIG. 21. Composite illustration showing sites of Ewing's tumors met with at Percy Jones General Hospital since January 15, 1943. The right hand illustration shows the other types of osteogenic tumors encountered. It will be noted that Ewing's sarcoma often involves a relatively long area of the shaft or may cause slight widening of the shaft simulating a nonsuppurative osteomyelitis. Insert on left shows metastatic mediastinal Ewing's tumor which arose from a soft tissue mass adjacent to the left internal tibial condyle (cumm. 7×2.5). Areas marked with small white centers indicate metastatic lesions in bones, liver and skull. Insert on right shows huge metastatic lesion involving left hemithorax mimicking a hydrothorax. Note the apparent predilection of the osteogenic tumors for the distal femoral and proximal tibial condyles. In our series we have not yet encountered Ewing's tumor of the distal femoral condyle.

sarcoma and a distinct aid in determining the time and site of biopsy.

3. Reference to Figure 21 will show the areas of skeletal involvement in most of the tumors of bone encountered at Percy Jones General Hospital during the preceding two and a half years.

4. Repeated roentgenographic examinations are essential in any case with symptoms suggesting early bone sarcoma. Small lytic lesions and slight periosteal reactions associated with tumors of the soft parts should be regarded with concern in soldiers in the second and third decades. In suspected cases a therapeutic test of roentgen irradiation may be invaluable.

5. Judicious deep roentgen therapy is a useful adjunct to surgical treatment in the period prior to operation when the clinical findings are being reviewed and permission is being obtained for operation.

6. Regardless of the form of treatment, the prognosis in Ewing's tumor is grave. Six of ten of our patients are dead or dying

on an average of nineteen months after the onset of symptoms.

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FIBROUS DYSPLASIA OF BONE*

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THE disease entity, fibrous dysplasia of the skeleton, has been generally recognized only since 1937. This recognition was, for the most part, the result of contributions to the literature by Albright and co-workers[1] and Lichtenstein.[5] Furst and Shapiro[3] showed in their review of the literature that this disease was known as early as 1922 but was described under various other titles such as "osteitis fibrosa cystica congenita" "precocious puberty and bone brittleness," and so forth. A survey of reported cases of fibrous dysplasia of bone was made by Lichtenstein and Jaffe[6] in 1942. They found that 75 cases of this disease were recorded in the literature. To this number, they added an additional 15 cases. Recently Dockerty and associates[2] described a series of cases all of which had bone lesions and extraskeletal changes including cutaneous pigmentations, somatic precocity in both sexes, and gonadal dysfunction in females. In their review of the literature, they did not include cases with only bone lesions and thus divided this disease into two types. In the case to be described no extraskeletal lesions were found.

The etiology of this disease is obscure. The prevailing opinion is that it is due to a developmental defect. It is characteristically discovered in childhood or early adolescence. A pathological fracture may be the first indication of the disease, or a limp with pain or deformity of the lower limbs may be the chief complaint. Facial asymmetry, ocular proptosis, and acromegalic changes may occur at times. The extraskeletal abnormalities are pigmentation of the skin; sexual precocity and endocrine dysfunction in female patients mainly, and hyperthyroidism. Dockerty and associates believe that somatic precocity may occur in both sexes.

Laboratory examinations are frequently negative in this disease. The blood phosphatase, however, may be elevated. In our case, we found a consistently elevated blood urea ranging between 44 and 48 mg. per 100 cc. However, we do not know whether this is of significance in relation to this disease.

One or many of the bones may be involved. Roentgenological studies, as Lichtenstein and Jaffe[6] point out, show the distribution of bone lesions to be predominantly unilateral. The characteristic lesions are scattered areas of rarefaction which have the appearance of bone cysts varying in size and shape. However, areas of increased density and overgrowth of bone are also found. Roentgen examination of lesions of the long bones show the cortex to be thinned and the medullary space to be distended. Frequently bowing of the long bones and coxa vara are present. Albright, in discussing Kornblum's paper[4] states that the roentgen examination seldom, if ever, shows involvement of the epiphyses in this disease. However, Dockerty and associates believe that early epiphyseal union and advanced bone age are found at times. In this patient the epiphyseal development and absence of epiphyseal union indicate normal bone age.

The main pathological findings in this disease have been well described by Lichtenstein and Jaffe.[6] The findings in our case are similar to theirs and those of other reported cases. Although the histopathological picture is not entirely characteristic, a biopsy is of aid and should be performed, if possible, in all cases where this disease is suspected.

It seems evident that although fibrous

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dysplasia has been looked upon as a cystic bone disease, cysts if present are an insignificant and secondary part of the lesion. The rarefied areas in the bone are ordinarily solid lesions, composed of collagenous connective tissue in which there may be small foci of ossification. In the main, the areas of bone show little osteoblastic activity. They are thought to represent metaplasia from the connective tissue. The calcification may be irregular, but in our case was fairly uniform. Occasionally, metaplastic foci of cartilage have also been found. Cysts, when present, are apparently the result of degeneration, necrosis or hemorrhage. Only insignificant numbers of lymphocytes are present; consequently the lesions are evidently not due to inflammation. No evidence of neoplasm has been found in any of the cases.

Fibrous dysplasia of bone has been found to be a self-limited disease. The activity of the disease process decreases with advancing age and is quiescent in adults. There is no therapy indicated in this disease except occasional bone curettage or rib resection of monostotic lesions. It may rarely be advisable to perform massive autogenous bone grafts to strengthen long bones which have undergone pathological fractures or are in danger of doing so. Generally, however, conservative treatment of these cases with proper caution against trauma and severe exercise in an effort to avoid spontaneous fractures is advisable.

CASE REPORT*

W. L., male, white, aged eleven, was admitted to St. Alexis Hospital on May 11, 1944, because of a limp which was noticed by his gymnasium instructor about February 1, 1944. The boy was well until four years ago, at which time he fell from a garage roof and suffered a fracture of the shaft of the right femur. He was in the hospital for thirty-five days and wore a cast for seven weeks. Following this fracture, he developed a slight limp which became progressively worse. Two years ago, he fell and fractured his right forearm and one year ago fractured the left forearm. Past history revealed that the child had the usual childhood diseases, including measles, chickenpox, whooping cough, and scarlet fever. There was no familial history of bone disease. The boy's paternal grandfather died of pulmonary tuberculosis. The father and mother are alive and well. The patient's two year old brother has had no serious illnesses. The developmental history disclosed that the patient was a full term normal infant weighing 5 pounds 4 ounces at birth. The Wassermann reaction was negative at birth. He was breast fed for three and a half months, and then alternated on S.M.A., evaporated milk, and grade A milk. He received no liver, vegetables, or juices until four years of age and was never given cod liver oil. He received ample air and sunlight. He was always undernourished and short of stature for his age. The mental development was normal; he sat up at nine months, stood up at ten months, and walked and talked at the age of one year. Smallpox vaccination and diphtheria toxoid inoculation were done when the boy was fifteen months old. Mantoux and Schick tests were negative at six years of age.

Physical examination revealed a fairly well developed boy who appeared to be about nine years of age and walked with a limp. He measured 4 feet 5½ inches in height. The parietal bones of the skull were more prominent than usual. The eyes, ears, nose, and throat showed nothing abnormal. The chest was normal in shape and there was good expansion. The lungs were clear to percussion and auscultation. The heart was normal in size, rhythm was regular, and the rate was 80 per minute. No abnormal cardiac sounds were heard. There were neither palpable masses nor tenderness in the abdomen. There was no pain or limitation of motion in any of the extremities. The left leg showed some atrophy of the thigh and measured 66 cm. from the anterior superior spine to the internal malleolus. The right leg measured 67 cm. using the same topographical landmarks.

Laboratory Findings. The urine revealed nothing abnormal. Urine examination for Bence-Jones protein was negative. There was no increase in urinary calcium excretion. Examination of the blood revealed the erythrocyte count to be 4,890,000, with 12.8 grams of hemoglobin per 100 cc. The leukocyte count

* We wish to thank Dr. R. J. Stasny for permission to report this case.

Fig. 2. May 13, 1944. Typical lesions of bones of the skull, involving especially the bones of the left side of the vault and base.

was 8,200, with 66 per cent polymorphonuclears, 32 per cent lymphocytes, and 2 per cent eosinophiles. The sedimentation rate was 5 mm. per hour. The blood sugar was 106 mg. per 100 cc. Repeated blood urea determinations varied from 43 to 48 mg. per 100 cc., and the creatinine ranged from 2.4 to 3.55 mg. per 100 cc. The

Fig. 1. May 13, 1944. Lateral view of skull showing thickening of occipital and basal bones and focal rarefied areas.

blood phosphatase was 5.28 Bodansky units. A blood Kline test was negative.

Roentgenologic Findings. The skull had a most unusual appearance (Fig. 1 and 2). There was marked thickening of the occipital and left parietal bones. The bones of the base of the skull in the anterior and middle fossa were thickened and showed marked increase in their density. There were numerous small, round, sharply defined areas of decreased density in the bones of the vault of the skull. The clinoid processes of the sella turcica were large and dense, especially the anterior. There was

Fig. 3. July 9, 1940. Pathological fracture of the right femur through the lesion at the site of fracture. Second lesion in upper fragment of the femur above the site of fracture.

FIG. 4. April 23, 1944. Typical lesions involving the bones of the pelvis and upper portion of both femurs.

sclerosis of the left mastoid process. The right femur (Fig. 3) had a healed pathological fracture of the middle third of the shaft of the femur through a small cyst-like area. (Fig. 3 shows the original fracture with rarefied area.) There was a larger rarefied area about an inch and a half long and three-quarters of an inch wide in the upper third of the shaft of the femur just below the level of the lesser trochanter. The medullary space here was widened and the cortex was thin. The pelvis (Fig. 4) contained numerous areas of decreased density most marked on the left, which appeared cystic.

FIG. 5. May 16, 1944. Typical lesions involving the bones of the left leg.

FIG. 6. Photomicrograph (×134) showing typical area from tibia. The bony trabeculae are fairly prominent. The fibrous tissue shows various degrees of cellularity.

The left side of the pelvis was slightly deformed and deviated a little toward the midline. There was decreased density in the neck and upper portion of the left femur. The upper end of the left femur showed marked deformity with lateral bowing of the shaft. The chest showed slight enlargement of the right fifth and left fifth and seventh ribs. In the left leg (Fig. 5) there were marked changes in the bone structure of the tibia, fibula and os calcis. There were areas of decreased density throughout the shaft of the tibia and in the lower two-thirds of the shaft of the fibula. There was moderate widening of the shaft of the tibia and fibula in the involved regions and the cortex was thin.

Pathological Findings. A biopsy was taken from the shaft of the left tibia. No cyst was identified. The gross specimen consisted of irregular bony fragments varying from 2 to 10 mm. in diameter. Most of the fragments were porous resembling cancellous bone, but the largest contained some dense, bony tissue resembling cortex. The tissue was fixed in Zenker's fluid.

Histologic sections revealed abnormal bony tissue and no distinction as to compact or cancellous layers could be made. No regular concentric bony lamellae were seen. The basic structure was collagenous connective tissue in which islands of bone were present. These varied in size and contour, and were irregularly distributed in the stroma. Although there was variation in the amount of bone, no areas were found in which the bone occupied less than approximately one-fifth of the total sectioned surface. For the most part, the masses were well calcified. The lacunar cells were of average appearance. There were occasional osteoblasts at the periphery of the masses but only few osteoclasts were noted. The stroma was composed of collagenous connective tissue in which the nuclei were considerable in number. Most of the nuclei were oval and vesicular, but others were narrow and dense. There was moderate vascularity. The capillary walls were thin and the lumens were filled with blood. The extravascular blood cells were presumed to be artefacts. Only an occasional lymphocyte and plasma cell was noted in the stroma. No cartilage was identified (Fig. 6).*

SUMMARY

A case of fibrous dysplasia of bone showing polyostotic involvement and unilateral thickening of the skull has been presented. There were no extraskeletal abnormalities. The clinical, roentgenographic and histopathologic aspects of this disease are discussed.

It has been shown that there are four salient features of this disease which must be regarded as the criteria for diagnosis. They are as follows:

1. The clinical syndrome manifested by onset of symptoms in childhood with ultimate local pain, deformity and disability of some extremity.

2. Roentgenographic evidence showing characteristic skeletal involvement of one or many bones, with a strong tendency to be unilateral.

3. Blood chemistry and urine studies which show absence of the results characteristic of other disease of the skeleton. The blood phosphatase is often moderately elevated.

4. Bone biopsy which shows a consistent histopathological picture.

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* We wish to acknowledge that the roentgenograms and histologic preparations were examined by Drs. L. Lichtenstein and H. L. Jaffe who concurred in our diagnosis.

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PARA-ARTICULAR CALCIFICATION IN THE LOWER EXTREMITIES OF PARAPLEGIC PATIENTS*†

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CALCIFICATION about the joints and in the soft tissues of the lower extremities of paraplegic patients has been reported rather infrequently. Derra and Nadermann[1] recently reported 2 cases and discussed the incidence and etiology. They reviewed many of the previous reports, mainly from the European literature, and found that the condition had been present in many of the veterans of World War I who had sustained spinal cord injuries. Some of the reports cited by these writers gave the incidence as high as 50 per cent for soft tissue calcification in the lower extremities of paraplegics. The etiology was admittedly uncertain, however they believed that it was in some manner related to irritative neural impulses following spinal cord injuries.

At this hospital 88 patients with spinal cord and cauda equina injuries of varying severity were observed during a two year period. Many of the early cases were retained at this installation only a short time and roentgenograms of the lower extremities were not obtained. Thirty-three patients were observed over a longer period of time; para-articular and soft tissue calcification in the lower extremities was observed in 4 cases. Only those cases that had swelling or stiffness of the joints were completely studied by roentgenograms, so the true incidence cannot be estimated. Three of these cases had only a minor degree of calcification in the soft tissues about the knee joints; the fourth case, however, had very extensive calcification about the knees and in the soft tissues of the lower thighs, and is presented here in detail.

CASE REPORT

A twenty-six year old American soldier sustained a small shell fragment wound of the spine at the level of the tenth thoracic vertebra on November 18, 1944. On November 19, 1944, a lumbar puncture was done below the site of injury and revealed a complete block of spinal fluid. Laminectomy was not done. During the month of December, 1944, while being evacuated to the United States, the patient rapidly lost weight and developed large decubitus ulcerations over both hips and the sacrum. He developed massive edema of both lower extremities about one week after injury. He arrived at this hospital on January 17, 1945. There had been no improvement in paralysis or anesthesia below the level of injury.

Physical examination on admission to this hospital revealed an extremely pale and chronically ill patient. There were large decubitus ulcers over both hips, heels, and the sacrum. There was a small well healed wound, measuring 1 cm. in diameter, just to the left of the spinous process of the tenth thoracic vertebra. There was complete anesthesia and flaccid paralysis below the eleventh thoracic segment of the spinal cord. There was marked pitting edema of both lower extremities from the toes to the groins. The red bood cell count was 2 million; the hemoglobin was 6.5 grams per 100 cc.

On January 31, 1945, roentgenograms of the lower extremities revealed extensive calcification in the soft tissue about both knee joints (Fig. 1). On February 24, 1945, a lumbar puncture revealed a complete manometric block. Supportive treatment with transfusions and proper diet resulted in marked improvement in the patient's general condition. In March, 1945, the edema of the lower extremities was negligible; a roentgenogram at this time revealed further extension of the calcification about the knees (Fig. 2). During April, 1945, the patient developed an extension of the infection from the decubitus ulcer over the left hip into the left thigh, and this required incision and drainage. During the next few months the patient's general condition rapidly improved and he was able

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to be up in a wheel chair. There was only 30 degrees of motion at the knee joints, and leg supports were required. In August, 1945, a skin graft was applied to the sacral ulceration which resulted in complete healing. By October, 1945, the decubitus ulcerations over the trochanters were healed; roentgenograms of the right hip revealed some fragmentation of the articulating surface of the head of the femur with marked para-articular calcification (Fig. 3). Fine granular deposits of calcium had been noted about this joint in May, 1945. Roentgenograms of the knees revealed essentially the same amount of calcification as had been noted in March, 1945.

Fig. 1. Roentgenogram of the right and left knees approximately two and a half months after injury. Para-articular calcification is already marked.

The patient was transferred to another hospital in February, 1946, at which time his general condition was excellent and he was beginning the use of braces and crutches for ambulation.

The consensus is that the calcification about the joints and in the soft tissues usually begins during the first year after the spinal cord injury, if it is to develop. In the cases studied, those that did not have calcification about the joints when roentgenograms were taken six months after injury did not subsequently develop any significant amount of soft tissue calcification, except in those cases that had late associated fractures. This extensive para-articular calcification associated with late fractures of the neck of the femur has been observed in 2 of the paraplegic

Fig. 2. Roentgenograms of right and left knees approximately four and a half months after injury. Increased calcification is evident.

patients. In the case reported in detail, it was noted following fragmentation of the

Fig. 3. Roentgenogram of right hip showing para-articular calcification. Fragmentation of articulating surface and a large decubitus ulcer (now healed) over trochanter contributed to its development.

articulating surface of the head of the femur. Lesser amounts of calcification in the soft tissue about the upper end of the femur and hip joints have been observed in those cases that had extensive decubitus ulcers over the trochanter.

The cause of the calcification is not certain. It is generally known that calcification occurs not infrequently in muscles adjacent to fractures, hematomas, areas of fatty necrosis and in irritated bursae. Many theories have been advanced to explain the development of this calcification. Piatt[3] observed calcification about the ankle joint and believed that multiple repeated minor traumatisms were predisposing factors. Nachlas and Olpp[2] in an investigation of para-articular calcification (Pellegrini-Stieda's disease) about the knee believe that it begins as the result of a series of small traumas which do not attract much attention and is followed by degenerative changes in the gliding membrane over the adductor tubercle. Minor incomplete lacerations occur in the altered tissues with small localized areas of bleeding, which under proper physical and chemical conditions lead to calcification. The stiffness and fixation of connective tissue structures about joints following immobilization is frequently observed and is accentuated when edema is or has been present. The paraplegic patient may present several conditions that predispose to the development of soft tissue calcification in the lower extremities. Edema of the lower extremities is a fairly frequent finding shortly after a spinal cord injury and is accounted for by several factors which include immobilization, vasomotor relaxation and nutritional hypoproteinemia. At times the edema may be massive and the lower extremities are swollen tense with a shiny pale surface. Persistence of the edema leads to fixation of organic compounds in the connective tissue structures rendering them susceptible to calcifications, or to repeated trauma. The process of attrition in the connective tissue structures about the joints may be accentuated by the slowing of venous return from the extremities incident to the flaccid paralysis.

The cases that have been observed to have even minor degrees of soft tissue and para-articular calcification have had massive edema of the lower extremities at some time after the spinal cord injury. These patients have usually had a complete flaccid paralysis. Shortly after the spinal cord injury there is a rapid demineralization of the bones of the lower extremities associated with a hypercalcinuria. It is difficult to know what part this rapid movement of calcium plays in the development of the soft tissue calcification, but it would appear that if predisposing conditions of the soft tissues are present the incidence and degree of its occurrence might be influenced by the presence of large amounts of mobilized calcium from the bones. The addition of repeated minor traumas to the connective tissue structures with degenerative changes may well result in further attrition and localized areas of hemorrhages, leading to calcification. The calcification about areas of fractures that have occurred late after injury is certainly related to trauma and hemorrhage into the soft tissue.

The lesser degrees of para-articular calcification (Fig. 4 and 5) do not interfere with the rehabilitation of the paraplegic patients. On the other hand, extensive calcification about the knees may so limit flexion that these patients are not able to sit up in a wheel chair or on the side of the bed unless supports for the legs are provided. Such patients have difficulty in dressing themselves and cannot use the ordinary folding wheel chair on which the leg supports are not adjustable. Calcifications about the hip joints are subjected to repeated trauma and may eventually limit motion to such an extent that sitting up is difficult. The extensive calcification and deformity that results from a fracture of the neck of the femur may prevent satisfactory rehabilitation. One patient has been ob-

Fig. 4. Lesser degree of soft tissue calcification along medial epicondyle in a patient with paraplegia.

Fig. 5. Lateral view of the knee shown in Figure 4 with calcification in patella tendon.

served who sustained a fracture of the neck of the femur when turning in bed. The fracture was not recognized for several weeks; the extensive calcification and deformity that has resulted prevents the patient from sitting up or beginning the use of braces. An extensive calcification about the knees is compatible with use of braces for ambulation.

There is no treatment recommended for extensive soft tissue and para-articular calcification. Prevention of fractures or chipping of articular surfaces resulting from too vigorous efforts at rehabilitation should be emphasized.

At installations where large numbers of paraplegic patients are to be cared for a study of the incidence and etiological factors may yield further information on this condition.

SUMMARY

1. A case of extensive para-articular calcification in the lower extremities of a paraplegic patient has been presented.

2. A short discussion of possible etiological factors is given.

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REVISED "OSSIFICATION INDEX" FOR THE DETECTION OF ENDOCRINE DISORDERS IN CHILDHOOD*

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THE use of an "ossification index" has been proposed in an effort to provide a quick, convenient and graphic scheme for indexing the advance of carpal ossification visualized in a roentgenogram taken for the study of skeletal progress in a growing child.[4] As explained in the original presentation, gross divergence from the normal may be thereby promptly detected and completely recorded. It is admittedly true that the entire answer to any question of "bone age" or "rate of ossification" will have to be sought in studies beyond the limited inspection of the region of the wrist. It is also quite possible that an index based upon an assignment of figures for the approximate *average* rather than the *latest limit* of age for normal appearance would have considerable value. It has, nevertheless, been thought best to present the *standard* "ossification index" as the one based upon figures which represent the ages for the latest normal appearance and to rely upon the suggested, more or less automatic interpretation to answer the most frequently asked question—whether or not the start of ossification in the bone centers of the specific child has been abnormally delayed.

ROUTINE USE OF THE "OSSIFICATION INDEX"

When the figures are posted from a roentgenogram and a center is found absent whose assigned figure is less than the actual age of the patient, the validity of the automatic conclusion that abnormal delay in the ossification has occurred depends upon the correctness of the ages chosen for the limits of normal. As stated, these figures have been obtained from a tabulation of ages quoted from as many studies as could be readily found in the literature.[4] Even so, it is suspected that some of these writers are quoting other authors so that the actual number of independent observations of the normal may be more limited than it seems. However, there is a general agreement among these authorities, and, for us, the use of the set of figures chosen by inspection of this tabulation has shown, with a single exception, that the originally proposed standard "ossification index" correctly expresses for each bone the true outside limit of slow normal. The observation of the one exception has resulted in a change of the age limit or appearance of ossification in the distal ulnar epiphysis from the seventh to the eighth year. The standard "ossification index" for the full complement of carpal bones at the age of eight has therefore been revised to read 8-67-2456778 (Fig. 1).

Posting of the actual "ossification index" for each child in these age groups admitted to the Mary McClellan Hospital for such things as appendectomy, tonsillectomy and orthopedic surgery as well as those seen in the weekly well-baby clinic has seemed a logical way to study the progress of ossification in a cross section of the child population (Fig. 2). For this study, a portable roentgen-ray unit was set up and kept ready so that the taking of the desired roentgenogram consumed less than a minute of clinic time (Fig. 3). Very useful was the improvised frame in which an 8 by 10 inch cardboard film holder was placed to mask one-half of the film and properly superimpose the identifying marker on the margin of the other half just above the patient's hand (Fig. 4). Cardboard film holders were chosen because the disadvantages of longer exposure seemed to be offset by the practicability of having quite a number of holders loaded and ready. A special felt-padded board was also contrived to hold the infant's hand against the

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OSSIFICATION INDEX*

For a rapid evaluation of the rate of ossification in a growing child a group of significant figures may be determined by x-ray and posted as an "Ossification Index." This proposal is made after considering two dogmatic short-cuts, one limiting the region of study in the belief that the wrist bones will reflect the rate of bone development in the rest of the body and the other adopting a set of arbitrary extremes computed from the published studies of the normal age of first appearance of the centers. To avoid confusion it is understood that a patient has not reached a specific age until he has completed that period of life.

NORMAL AGE EXTREMES FOR FIRST APPEARANCE OF OSSIFICATION CENTERS

Bone	Earliest	Majority	Latest
Capitate	Birth	2½ months	6 months
Hamate	Birth	3½ months	7 months
Radius (epiphysis)	6 months	1 year	2 years
Triquetrum	1 year	2¼ years	4 years
Lunate	2 years	4 years	5 years
Navicular	4 years	5 years	6 years
Lesser Multangular	4 years	5¼ years	7 years
Greater Multangular	4 years	5¼ years	7 years
Ulna (epiphysis)	5 years	6¾ years	8 years **

A diagramatic representation of the order in which the centers appear and the ages of the latest first sign of ossification within the normal limits will assist in the computation of the index. (See diagram on the reverse side of this card.) The index itself is constructed by setting down the patient's age followed by the figures for the centers actually present. These are placed in their chronological order and zeros substituted if any center has not yet appeared. For additional clearness a separation is made with hyphens between the figures representing months and those representing years. Thus the normal index for the full complement of ossification centers at the age of eight years becomes "8-67-2456778." Because the index is composed of figures representing the latest possible normal appearance date for the centers, most of them will be found to have appeared earlier. Many young patients will therefore show figures in the index greater than their actual age. When using the index to detect delayed ossification these extra figures will have no significance and the NORMAL OSSIFICATION INDEX may still be considered as a group of figures which includes all of the numbers assigned for the centers up to the actual age regardless of how many more may be present. Any index arrived at by inspection of the roentgenogram and found not to include all of the figures up to and including the actual age of the patient is abnormal and indicates a delay in ossification which is pathological.

* Leonard, D. W. Early Recognition of Endocrine Disorders in Childhood by Roentgenograms of the Wrist to Determine the "Ossification Index" Amer. Jour. Roentgenology, 53:55-61, Jan. 1945.

** Note: Figure for the age of latest normal appearance of the distal ulnar epiphyseal center has been revised.

Fig. 1. Facsimile of both sides of a card ($3\frac{1}{2}$ by $6\frac{1}{4}$ inches) prepared for convenient determination of the "ossification index" from any roentgenogram of the carpal bones as suggested in a previous publication.[4] Explanation of the method printed on the card includes a table of extremes for comparison.

film and with this it was found that a fairly satisfactory roentgenogram could be taken even with a struggling infant.

As the routine progressed and the graphic tabulation began, it was discovered that comparative results could only be obtained if the ages were expressed uniformly. For this reason it was decided that a child would be considered the age of his last birthday until he had completed another full year. The roentgen negatives of patients below the age of one year were marked by the number of weeks since birth and this figure was later converted into months according to the following schedule:

0 to 4 wk.	Condition at birth
5 to 8 wk.	1 mo. old
9 to 12 wk.	2 mo. old
13 to 16 wk.	3 mo. old
17 to 20 wk.	4 mo. old
21 to 25 wk.	5 mo. old
26 to 30 wk.	6 mo. old
31 to 34 wk.	7 mo. old
35 to 38 wk.	8 mo. old
39 to 43 wk.	9 mo. old
44 to 47 wk.	10 mo. old
48 to 51 wk.	11 mo. old.

By this study of the bone development in the wrist using the standard "ossification index" it has been sufficiently demonstrated that few clinically normal children will be incorrectly designated as abnormal in ossification. This study, therefore, becomes a successful test of the suggested "automatic interpretation" based upon the presence or absence of centers in relation to the actual age. Some objection may be

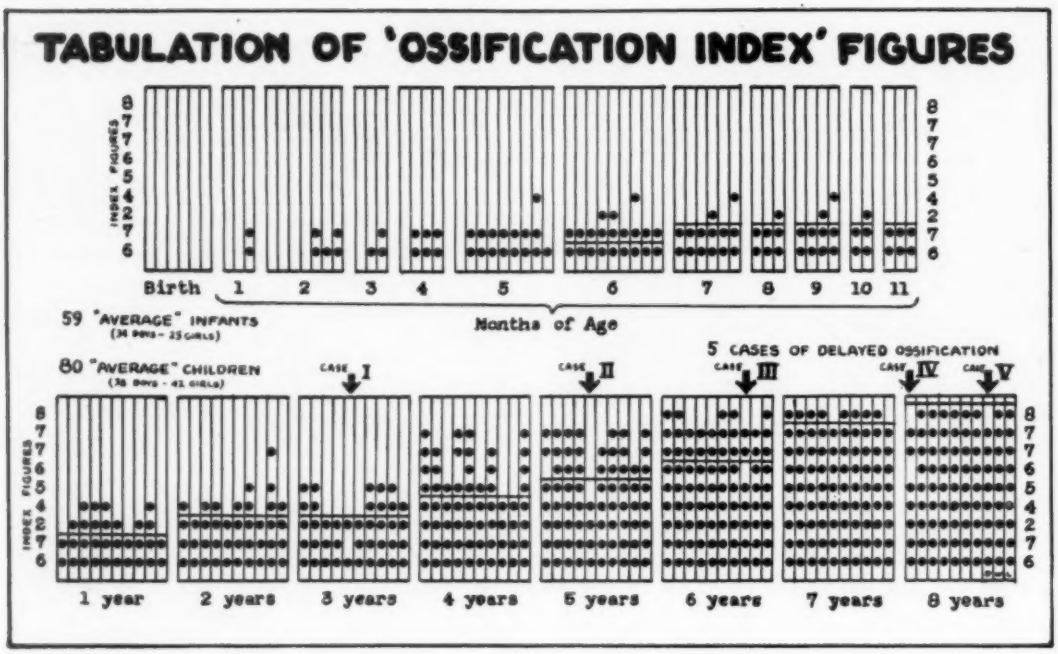

Fig. 2. Graphic summary obtained by plotting the actual "ossification index" for each of 59 infants and 80 children up to eight years of age. These indexes were obtained from average children without plan other than that of securing 10 of each age after the first year. The bones of the wrist have been numbered with the latest age of normal first appearance and plotted upward so that reading the vertical column in each case gives the "ossification index." The horizontal line in each age group indicates the level which should be reached at that age and all cases whose spaces are filled below that level are normal. Inspection of the result demonstrates 5 abnormal cases. These are all boys and may be described as follows:

Case I. W.E.S., male, aged three. Index: 3-60-2000000. Diagnosis: recurrent congenital clubfoot, bilateral. Ossification also delayed in feet. (Case included in series discussed *J. Pediat.*, 1945, *26*, 379.)

Case II. R.F., male, aged five. Index: 5-67-2400000. Diagnosis: subacute appendicitis. Operation done with normal recovery. No history of subnormal development.

Case III. E.C.R., male, aged six. Index: 6-67-2450770. Diagnosis: hypertrophied tonsils and adenoids. Operation done with normal recovery. No history of subnormal development.

Case IV. H.R.N., male, aged eight. Index: 8-67-2450770 (both wrists). Diagnosis: dislocation of epiphysis, right radius. Colle's fracture of left radius. Fractures reduced and immobilized. No history of subnormal development or evidence of pathological fracture.

Case V. G.E.S., male, aged eight. Index: 8-67-2456770 (right hand) 8-67-2456778 (left hand). Diagnosis: scar of burn and flexion deformity of right hand; hypertrophied tonsils and adenoids. Operation done to remove tonsils and a skin graft done on the right hand. No history of subnormal development.

raised to the practice of placing the latest age limits so far beyond the averages or to the expression of the ages themselves in round numbers. It may also be said that the limits have been pushed so far that borderline cases of delayed ossification will be missed. It will, nevertheless, have to be admitted that there is definite virtue in the fact that when delay *is* indicated by the "index," it can be accepted as unmistakably abnormal. By reference to a table of earliest and average as well as latest age of appearance, an "index" which is normal by the rule can still tell the examiner a good deal about minor variations, and if taken at the right moment (as would be necessary by any standard) the "index" can show disturbances in the normal *order* of appearance of the centers in the disharmonic development stressed by some writers. All of these things can be read from the figures of the "ossification index" in addition to the making of the survey for missing centers.

The data obtained by indexing the unselected cross section of the child population, as described, have been analyzed

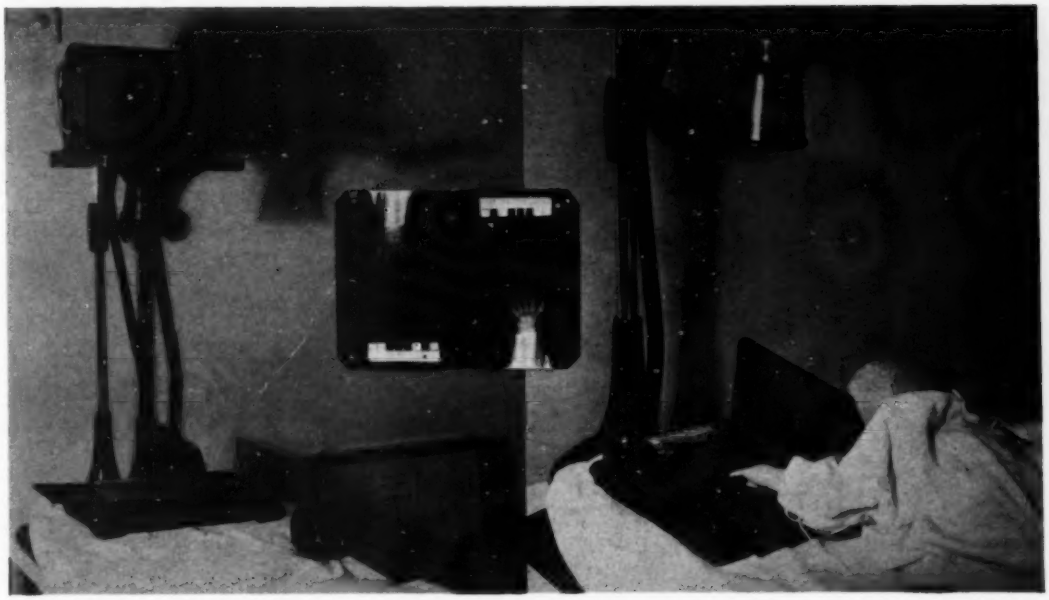

Fig. 3. Portable roentgen-ray unit set up for the clinic roentgen examinations necessary to determine the "ossification index." Cone on roentgen tube limits the field of exposure to a circle which just covers half of an 8 by 10 inch film. Centering is done by aligning the hand with the supporting post. An infant may be placed face down, as illustrated, with the head out of the field behind the upright portion of the film holder and older children may stand beside the table.

graphically (Fig. 2), and from the uniformity of the grouping it can reasonably be assumed that conclusions from this relatively small series are reliable.

Interpretation of the "Ossification Index"

From several sources has come the comment that the use of the word "index" for this listing of the ages assigned to the centers is misleading. The term, of course, has been correctly defined as a figure obtained by the reduction of a fraction representing the ratio between what is normal and what is actually present. At the same time the word "index" has also correctly been defined as a list of several items in an arrangement for easy reference. This last conception is the one which led to the introduction of the term "ossification index" and it is believed that no other designation will serve as well, even if it is necessary to admit that in the other sense it is not a true index.

The "ossification index" brings order and compactness to a subject which has always been vague in the average medical mind. With the "index" the roentgenologist can send an understandable report to the referring clinician and if the latter, through greater than average familiarity with the irregularities of early ossification, wishes to go beyond the automatic interpretation suggested, the "index" can still be found to contain much of the information necessary for his more refined analysis. The individual clinician may reconstruct a mental picture of the roentgenogram from which the "index" was taken and draw his independent conclusions regarding the applicability of these data in the wrist to the entire body of his patient. The use of the "ossification index" is suggested for those who write or speak of delay or precocity[3] of bone formation and wish a concise way of demonstrating the facts to the listener. That most of the disturbances of the ossification rate are due to endocrinopathy has been accepted, but the finding of delay graphically recorded in the "index" cannot by itself establish either the diagnosis of hypothyroid-

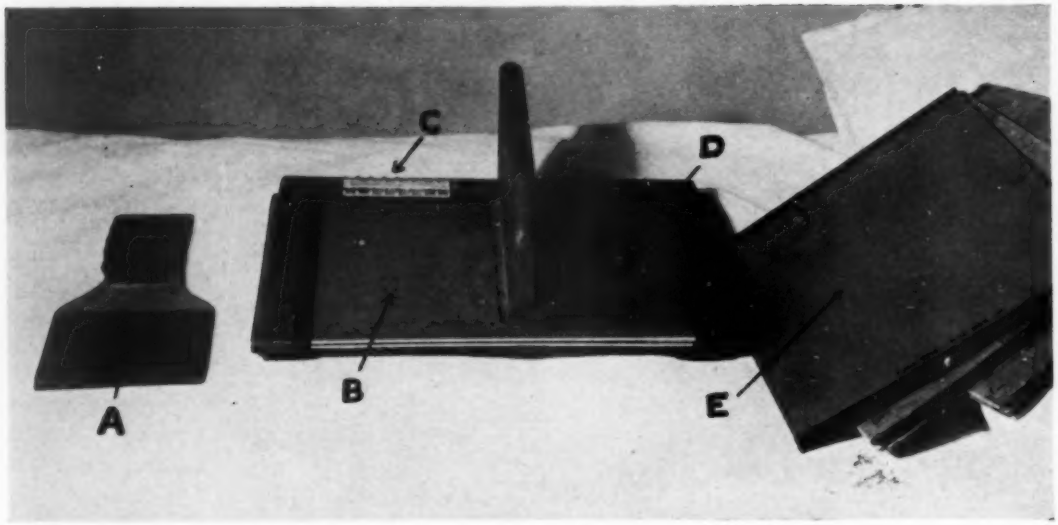

Fig. 4. Improvised holder for films to make possible the rapid taking of wrist roentgenogram at any age. Felt padded board (*A*) is hooked under the upright portion of the holder and pushed down onto the hand to hold it against the cardboard film holder (*B*) when fixation is necessary. The unused half of the film is covered by a lead shield (*D*) and the numbers placed in an attached film marker (*C*) record the age and identity of the patient. Film holder is withdrawn from the slot and changed end-for-end to expose the unused half while a supply of loaded holders (*E*) is ready in some suitable place for additional patients.

ism,[5,7] pituitary deficiency,[1,2] rickets[6] or dwarfism.[1,7] As always in such studies, the entire clinical picture must be considered. It is also a fact that the finding of normal ossification cannot be taken as excluding the possibility of acute endocrine dysfunction which has been present too short a time to affect the process. And yet, it can still be repeated that the demonstration of abnormal delay in bone formation through the use of the "ossification index" with the age figures chosen is always significant and a hunt for corroborating and differential signs such as the changes in the radius described by Woolley[8] should be instituted.

CONCLUSION

It is suggested that the general practitioner, the school doctor and the pediatrician employ the simple expedient of referring the subnormal child to the roentgenologist for roentgen-ray measurement offered by the "ossification index." This would be an easy initial step in the study of such a child.

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THE ROENTGEN AND CARDIAC MANIFESTATIONS OF FUNNEL CHEST*

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FUNNEL chest, or pectus excavatum, has received scant attention in the literature and textbooks. The first clinical description is credited to Johann Bauhinus, who in the sixteenth century, described a case with " . . . dyspnea, associated with cough increasing to paroxysms of suffocation."[8] Von Eggel, in 1870, described cases with cardiac embarrassment which he attributed to the deformity. A monograph of 21 case reports was published in 1904 by Gerstenberg. With the advent of roentgenography, the effect of this deformity upon the position and shape of the heart was noted by a number of observers (Verse, 1910; Stadtmuller, 1920; Sauerbruch, 1920; Meyer, 1911; von Hoffmeister, 1927; and Alexander, 1931).[3]

Figures 1 to 9 illustrate our 9 cases of funnel chest. The salient clinical, roentgeographic, and electrocardiographic features of each accompany the illustrations.

Funnel chest is not a rare condition. Since the greatest number of individuals having this deformity are without troublesome symptoms, only an occasional one will present a medical problem, and only a small number of these will be reported in the literature.

The malformation is a developmental anomaly in nearly all instances.[8] The defect, however, may be hereditary, as noted in Cases III, IV, V, and VII, where it was present in one or more members of the patient's immediate family. In severe rickets this type of deformity may sometimes result, although pigeon chest is a far more common finding. Obstruction to free respiration in children with forced respiratory movements has been considered as a possible etiology, but this seems unlikely. Severe trauma to the sternum may produce an angulated fracture with resultant funnel chest. This possibility is considered in one of our cases (Case VIII).

In the non-traumatic cases, there is usually no deformity present at birth. In the first years of life the sternal depression appears and gradually becomes increasingly more marked. Because it is absent at birth, the maldevelopment may be erroneously attributed to rickets, or obstructive respiration.

Funnel chest is due to a posterior position of the sternum with relation to the lower anterior chest wall. The manubrium is usually not involved, and the lower segment of the corpus sterni commonly forms the most posterior portion of the deformity.[3] The anterior ribs and costal cartilages maintain their normal position, leaving the anterior chest wall intact on both sides of the sternum and allowing a depression to be formed centrally. The deformity may be of varying degrees, with a depth reaching at times to 7 or more centimeters in severe cases. Not uncommonly, the depression is somewhat irregular, with the greatest depth not centrally placed.

The heart, lying beneath the sternum, is directly affected by the deformity. A displacement of the heart to the left is a fairly constant finding and was noted by a number of early observers.[3,8,20] Sometimes the

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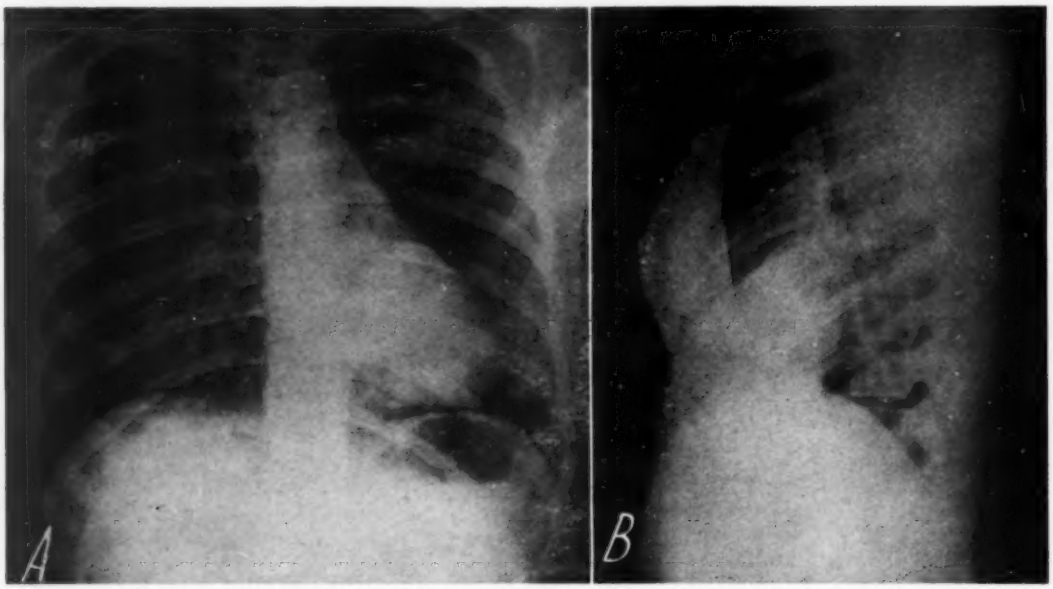


FIG. 1, *A* and *B*. Case I. F. W., clerk, aged eighteen. *Family history:* Not significant. *Past history:* "Growing pains" and acute tonsillitis as child. *Symptoms:* Palpitation on rest and exertion, dyspnea on exertion. Fair capacity for work. Faints easily. *Physical findings:* Blood pressure, 120/80; pulse at rest 80, after exertion 110, two minutes following exertion 96. Slight right upper quadrant abdominal tenderness. Maximum depression of the lower sternum 3.5 cm. *Electrocardiogram:* PR interval 0.20 sec., inverted T3. *Roentgen findings:* Funnel chest deformity of the lower sternum with shift of the heart to the left.

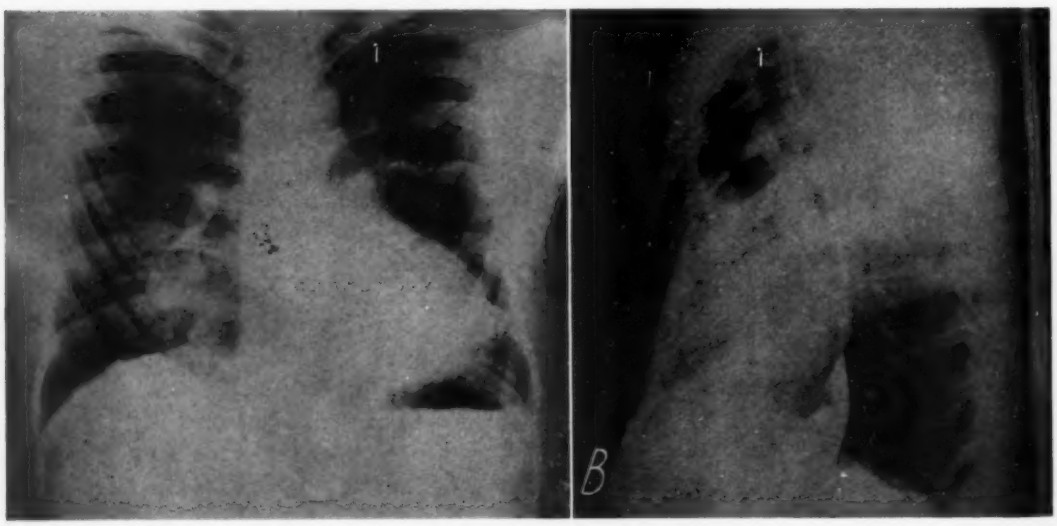


FIG. 2, *A* and *B*. Case II. W. M., economic analyst, aged thirty-two. *Family history:* Not significant. *Past history:* Scarlet fever followed by nephritis. *Symptoms:* Palpitation only when sleeping on the left side. Engages in strenuous exercise without ill effect. *Physical findings:* 1 + cyanosis of the nails, bigeminal rhythm. Blood pressure, 145/65. Maximum depression of the lower sternum 7 cm. Distance from the lowest point of depression to the ventral surface of the dorsal spine 6.5 cm. *Electrocardiogram:* See text for complete discussion. *Roentgen findings:* There is a marked concavity deformity of the anterior chest with the heart shifted to the left although it is not enlarged.

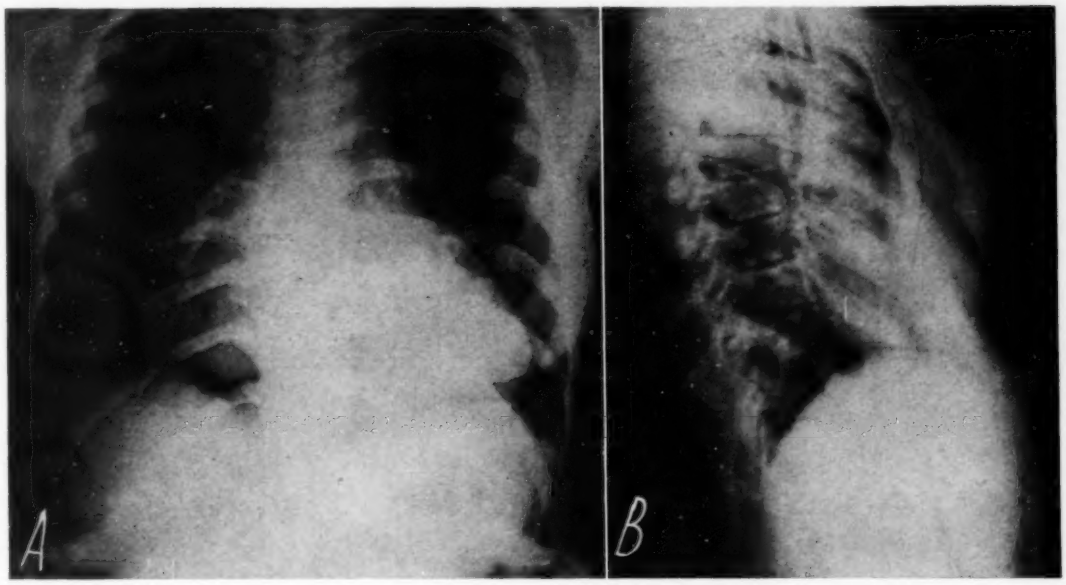


Fig. 3, *A* and *B*. Case III. W. F., clerk, aged seventeen. *Family history:* Father and brother have funnel chests. *Past history:* Not significant. *Symptoms:* "Shortwinded" with moderate exertion. *Physical findings:* Blood pressure 115/70; deformity extends almost entire length of the sternum and ends in a flaring of the lower costal margin. Maximum depression 3.5 cm. *Electrocardiogram:* Slight right axis deviation, notching of P_2 and P_3, inverted CF_4. *Roentgen findings:* Fairly marked funnel chest with displacement of the heart to the left.

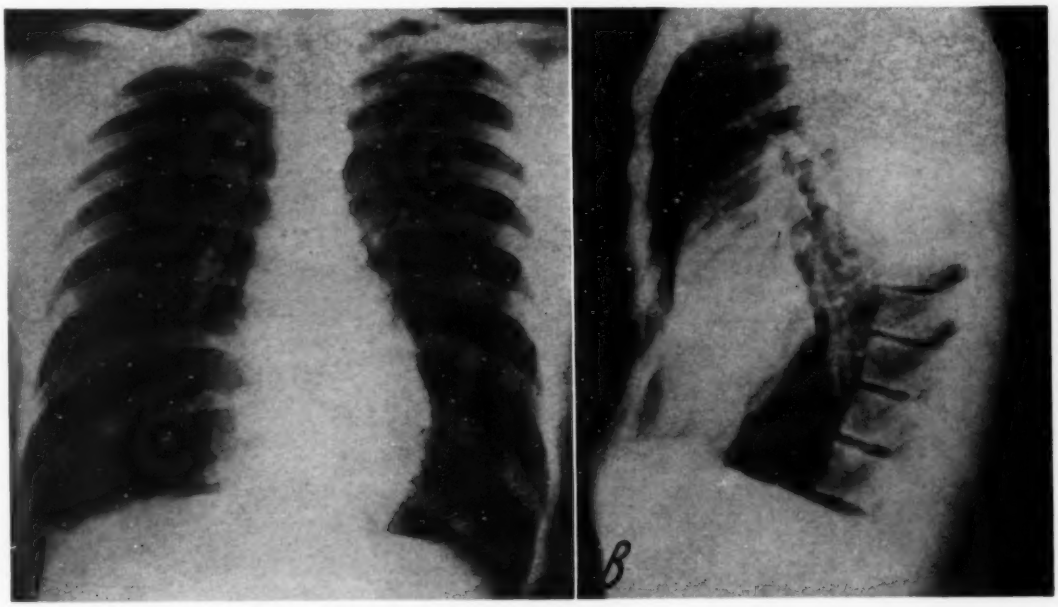


Fig. 4, *A* and *B*. Case IV. W. M., Maritime Service enrollee, aged twenty-two. *Family history:* Father has a slight funnel chest. *Past history:* Pneumonia, nephritis, tonsillitis. *Symptoms:* Palpitation with excitement and dyspnea with severe exertion, occasional episodes of "weakness" early in morning particularly in hot weather, mild chest pain when leaning to the left with the left arm outstretched; distress is localized in the left lower anterior chest and is definitely related to position changes—no anginal features. *Physical findings:* 1 + cyanosis of the nail beds. Blood pressure, 110/64, chest deformity is shallow and diffuse, measuring 9 × 11 × 3.5 cm. and extending upward to the level of the anterior third rib; left side of chest grossly more flattened than the right. *Electrocardiogram:* Not abnormal. *Roentgen findings:* Funnel chest deformity.

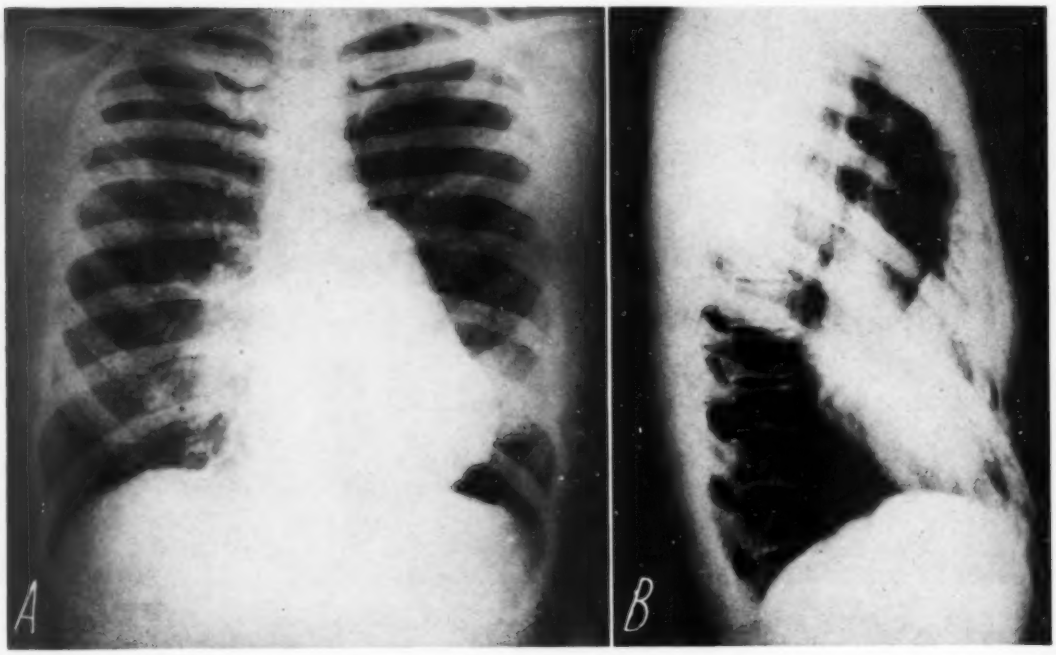


FIG. 5, *A* and *B*. Case V. W. F., social science analyst, aged thirty-one. *Family history:* Brother and daughter have funnel chests. *Past history:* Nephritis. *Symptoms:* Palpitation and dyspnea with moderate exertion, occasional ankle edema after standing for long periods. *Physical findings:* functional systolic murmur at the pulmonic area. Blood pressure, 110/70, maximum depression of the lower sternum 4 cm. *Electrocardiogram:* Inverted T in CF 4. *Roentgen findings:* Marked concavity deformity of the lower sternum. The heart is displaced to the left and shows a prominent pulmonary conus.

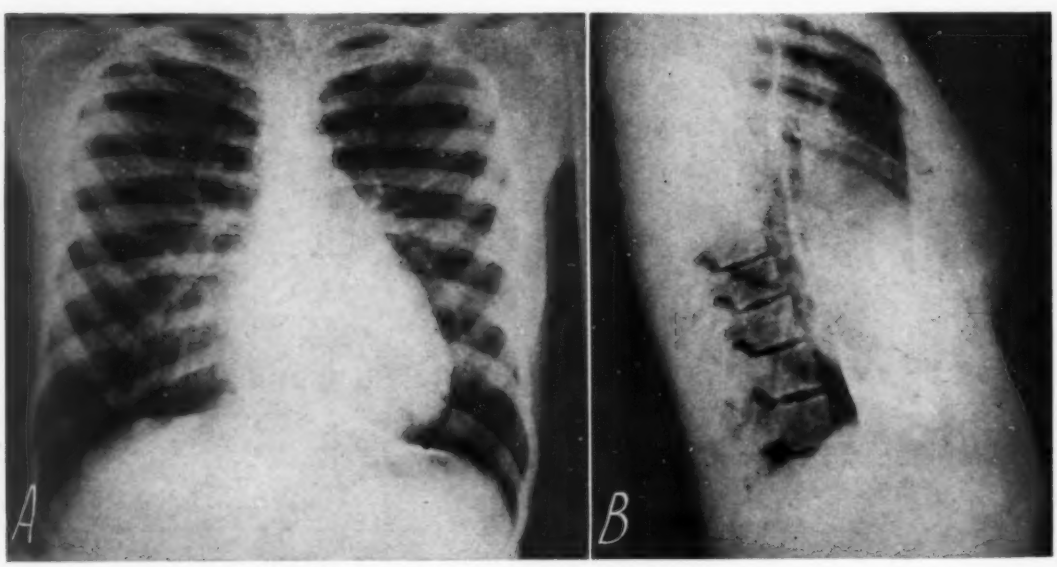


FIG. 6, *A* and *B*. Case VI. C. F., clerk, aged nineteen. *Family history:* Not significant. *Past history:* Influenza. *Symptoms:* Palpitation and dyspnea with moderate exertion, occasional attacks of dizziness with sudden changes in position. *Physical findings:* Blood pressure, 130/70, maximum depression of the lower sternum 3.5 cm. *Electrocardiogram:* Broad S wave in lead 2. *Roentgen findings:* There is a funnel chest deformity of the sternum with slight displacement of the heart to the left.

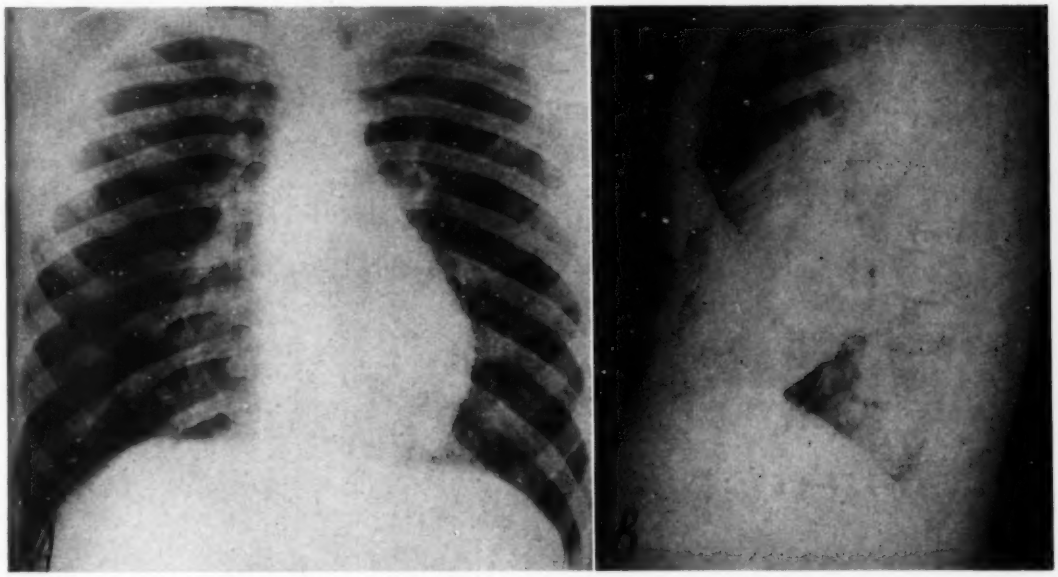


FIG. 7, *A* and *B*. Case VII. W. M., clerk, aged twenty-six. *Family history:* Brother has a funnel chest. *Past history:* Not significant. *Symptoms:* Intermittent pain in the left chest aggravated by coughing. *Physical findings:* Blood pressure, 110/65, slight hyperesthesia over the fourth and fifth anterior ribs in the midclavicular line, maximum depression of the lower sternum 4 cm. *Electrocardiogram:* Elevated ST segment in leads 1 and 2 (less than 1 mm.), slurred QRS in leads 2 and 3, low voltage T wave in lead CF 4. *Roentgen findings:* There is a funnel chest with slight displacement of the heart to the left. The heart is not enlarged.

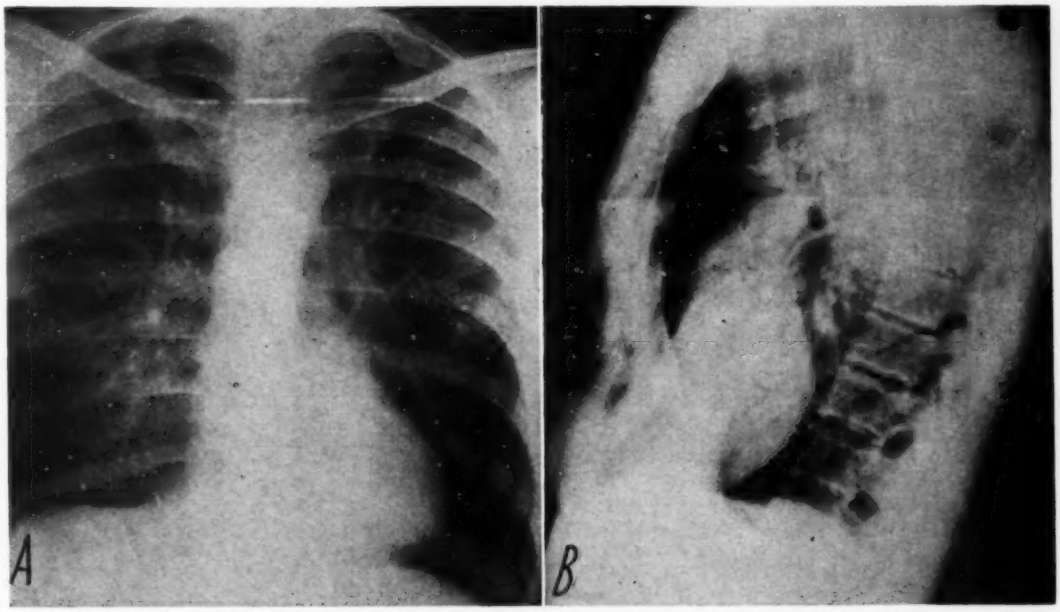


FIG. 8, *A* and *B*. Case VIII. W. M., cost accountant, aged fifty-one. *Family history:* Not significant. *Past history:* Influenza, pleurisy, scarlet fever, nephritis. Does not recall the presence of the deformity prior to the time he was kicked in the chest by a horse at the age of ten years. *Symptoms:* Dyspnea and palpitation on exertion, occasional bouts of ankle edema after standing for long periods, occasional fleeting chest pains along both costal margins; no anginal features. *Physical findings:* 1 + cyanosis of the nail beds. Blood pressure, 135/95, epigastric pulsation with loud blowing epigastric systolic murmur, maximum depression of the lower sternum 4.5 cm., minor varicosities of the lower extremities. *Electrocardiogram:* Slight elevation of the ST segment in leads 2 and 3, small R and deep S wave in lead CF 4, notched P wave in lead 2. *Roentgen findings:* Funnel chest deformity of the sternum with slight shift of the heart to the left. There is no evidence of old rib fracture to substantiate a traumatic etiology of the deformity.

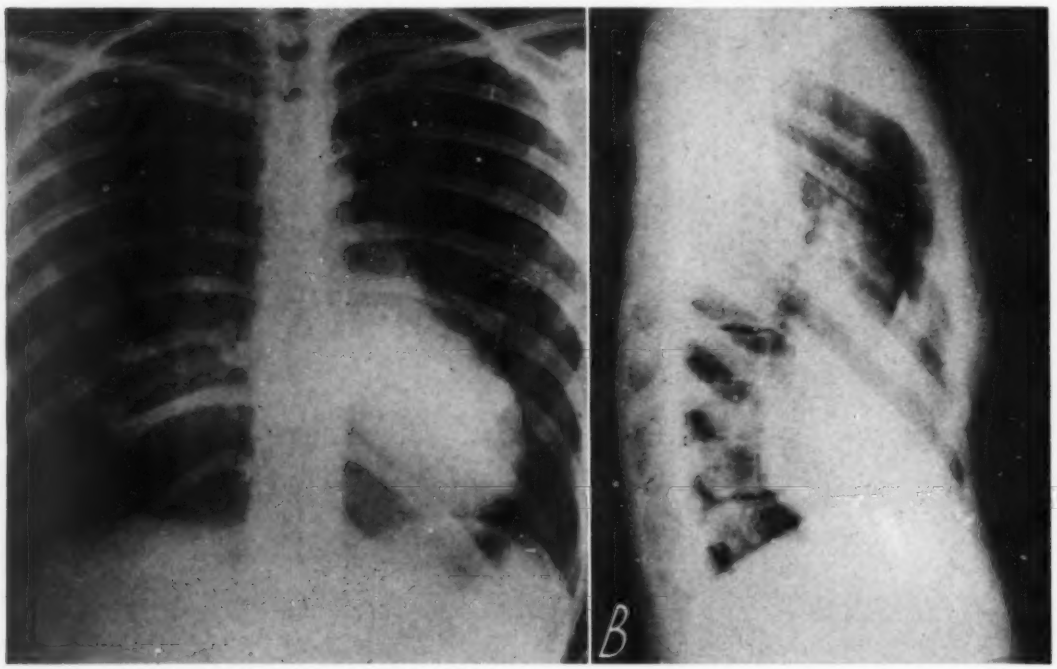


FIG. 9, *A* and *B*. Case IX. W. F., stenographer, aged twenty-nine. *Family history:* Not significant for other cases of funnel chest. Believes that two brothers have "heart disease" but does not know the type. *Past history:* Influenza. *Symptoms:* Palpitation at rest and with exertion, dyspnea on moderate exertion, edema of the ankles in hot weather, occasional pain in region of the second left thoracic interspace when fatigued, dull and somewhat constricting in character. *Physical findings:* Blood pressure, 110/70, second pulmonic wound louder than the aortic second, maximum depression of the lower sternum 3.5 cm. *Electrocardiogram:* Slight right axis deviation, slurring of the R wave in leads 2 and 3, low voltage of the T wave in lead CF 4. *Roentgen findings:* There is a moderate funnel chest deformity with a corresponding shift of the cardiac silhouette to the left. The heart is not enlarged. There is a wedge-shaped tenth dorsal vertebra which is probably congenital in origin.

cardiac displacement may be only slight but occasionally the entire heart may be so shifted as to be found lying in the left chest. Cardiac rotation in the sagittal plane may be present (Stadtmuller) but the heart is rarely widened in its lateral diameter.[3]

The roentgen findings are quite characteristic. In the posteroanterior view, the heart is shifted to the left, with some elevation of the left border from the diaphragm. This appearance is readily mistaken for a right ventricular enlargement, and since the right cardiac border is usually hidden by a vertebral column, an interpretation of cardiac enlargement could be made unless the true situation is realized (Fig. 1, 2, 3, 5 6, 7, 8 and 9). One of our patients (Case III) had been told a number of years ago that her heart was enlarged, and that she should adopt a conservative exercise program.

On lateral view, the diagnosis is self evident. The sternum protrudes backward, and is well behind the anterior chest wall (Fig. 1–9).

In examining photofluorograms of the chest it is important to be aware of the cardiac picture in funnel chest. Oftentimes, on the single posteroanterior roentgenogram, an erroneous interpretation of cardiac enlargement, congenital heart disease, or even mitral disease may be made. Even without a significant history or lateral roentgenogram, a presumptive diagnosis of funnel chest can be made when the heart is displaced to the left, with the right cardiac border hidden by the vertebral column, and with an elevated rounded left

lower cardiac border. This, of course, presupposes the absence of visible lesions which commonly cause a left cardiac shift, such as scoliosis, left pleural thickening, left-sided atelectasis, right-sided pleural fluid, tumors and other known conditions.

Of our 9 cases, all but 1 (Case IV) had a well marked displacement of the heart to the left. In this case, there was some pleural thickening in the right costophrenic angle which may have pulled the heart somewhat from its expected left-sided position. This is, of course, purely speculation.

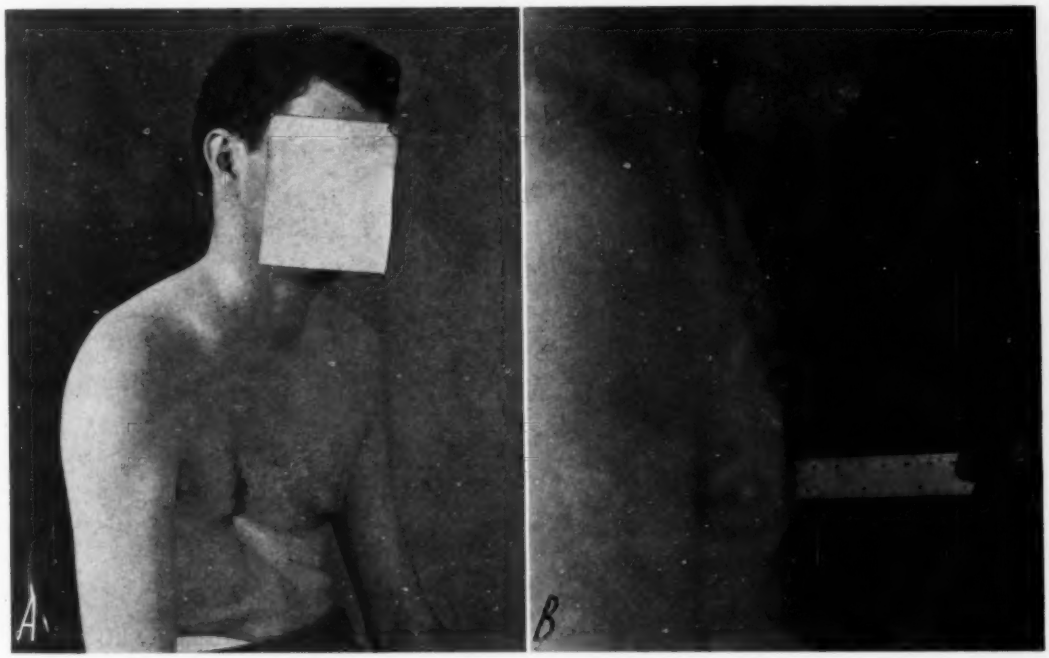


FIG. 10, *A* and *B*. Front and side view of Case II showing the marked depth (7 cm.) of the lower sternal depression. See Figures 2, 11, 12, 13, and the text for complete clinical discussion including roentgen and electrocardiographic findings.

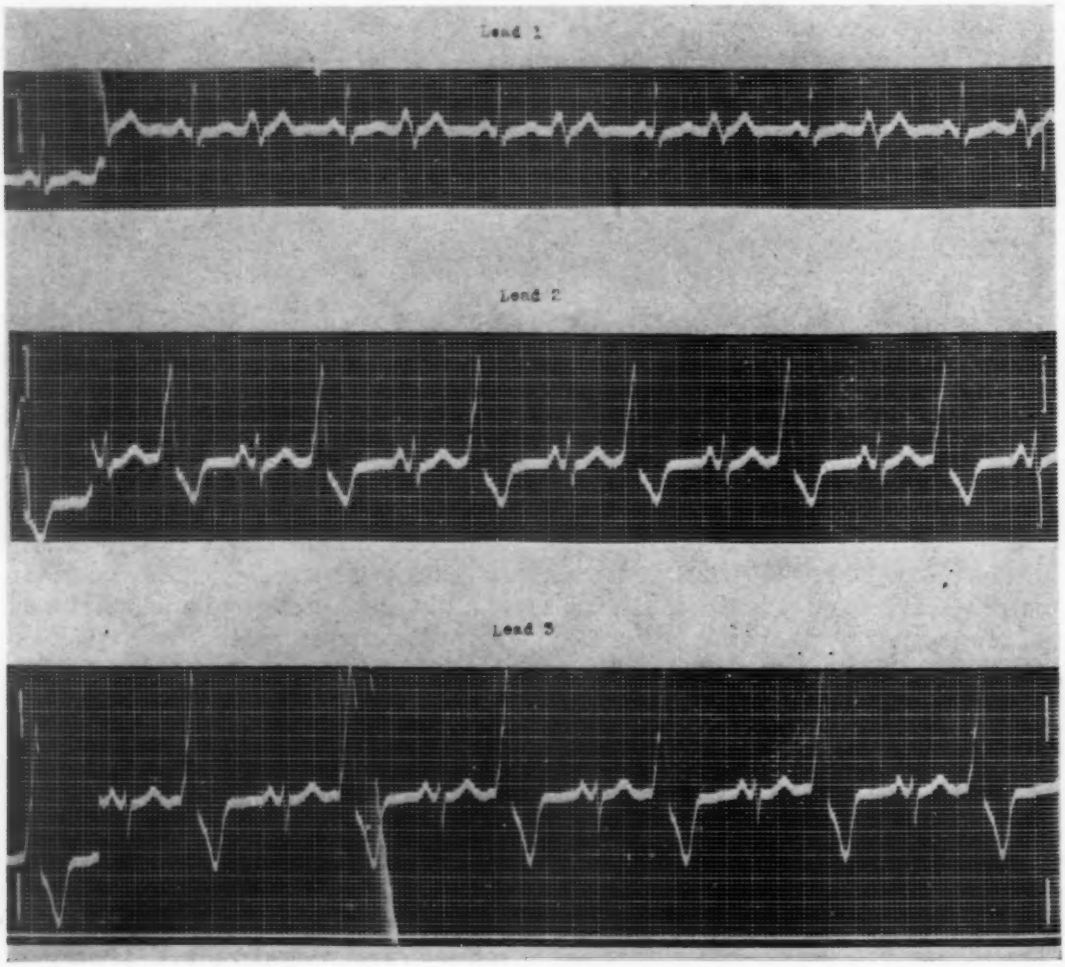
Another striking feature in a number of cases is the prominence of the right hilar and trunk shadows which are normally obscured by the overlying right cardiac border. This finding may at times be misinterpreted as an abnormal density in the right hilum and base resulting in an erroneous impression of pneumonitis or congestion.

The literature offers several instances of electrocardiographic abnormalities occurring in association with funnel chest. In Grieshaber's series right axis deviation occurred in 50 per cent (mild in 17 per cent, marked in 33 per cent). This finding was noted particularly with funnel chest of moderate and advanced degree with the heart lying to the left. Left axis deviation occurred in only 20 per cent of his series. P wave changes were encountered quite commonly and usually consisted of an enlargement of this complex most commonly in lead 2, and often associated with changes in the ST segment and T wave.[7] The PR interval was usually found to be unchanged. All varieties of T wave change including voltage elevation, peaking, particularly in lead 2 and less often in leads 1 and 3, and diphasic and negative complexes in leads 2 and 3 have been reported.[3,7,8] ST elevation was reported often by Grieshaber[7] but was of minimal degree and " . . . does not compare with that found in pericarditis and myocardial infarction." That author points out the necessity of recognizing

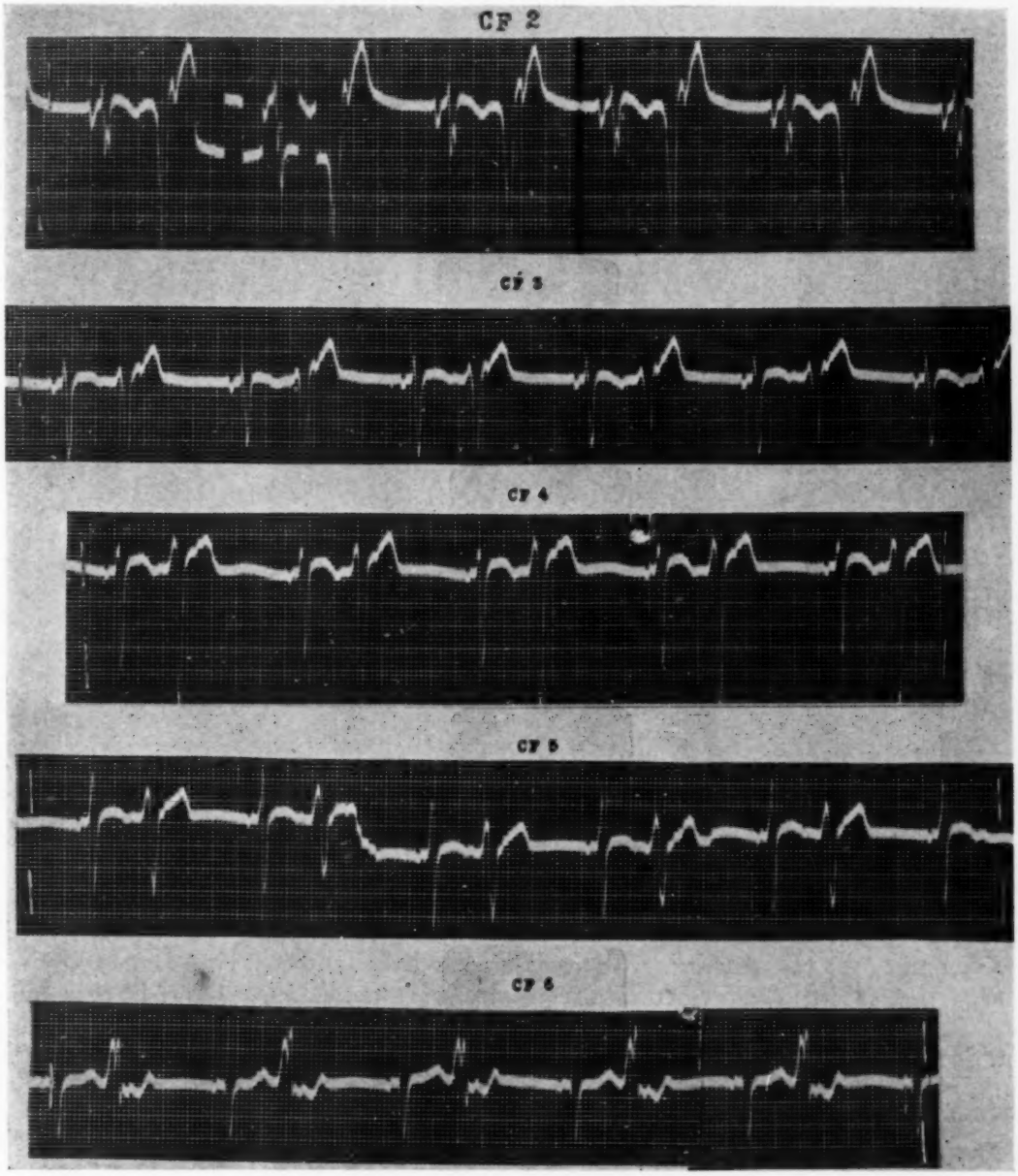
changes due to respiratory and extracardiac neurogenic influences. Alterations in his tracings taken after exercise were not remarkable and in only two instances did the T wave in lead I become smaller with a simultaneously unchanged T wave in leads 2 and 3, a pattern which he states is, " . . . more frequent with heart injury than with a sound condition."

The electrocardiographic changes in our series were of a relatively minor nature, as would be expected since none of the patients had any known cardiac pathology. Right axis deviation occurred twice (Cases III and IX) out of a total of 9 cases and in each instance was of a relatively slight degree. P wave changes were also less frequent than in Grieshaber's series, notching in leads 2 and 3 occurring in 1 case (Case III). No cases of large P waves previously described[7] were encountered. Minor ST segment elevations were found in 2 leads in each of 3 cases (Cases IV, VII and VIII) but in no case did the elevation exceed 1 mm. Notching and slurring of QRS was common especially in connection with S waves in the standard leads. A definite S wave occurred 5 times (Cases I, III, IV, VI and IX) and was deep in 2 instances (Cases III and IX). This finding was mentioned by Grieshaber as being an accompaniment of the cardiac rotation which commonly occurs in funnel chest.

To our knowledge, the electrocardiographic changes occurring in the chest leads in association with this condition



A



B

FIG. 11, *A* and *B*. Control tracing of Case II showing the standard (*A*) and CF chest leads (*B*). There is a bigeminal rhythm with regularly occurring right ventricular extrasystoles. Rate 100, PR interval .14 sec. in the sinus beats. QRS interval .07 sec. for the sinus and .12 sec. for the ectopic beats. Axis deviation, approximately 25 degrees. Principal initial deflection of the extrasystoles is upright in all leads. T waves of the sinus beats are inverted in leads CF_2, CF_3, and CF_4. T waves of the ectopic beats are opposite to the principal deflection of QRS except in lead 1, where the small total deflection of QRS suggests that this may be an "unfavorable" lead.

have not previously been described. Lead CF_4 was taken on each patient. In 2 instances (Cases II and VII) the ST segment was slightly elevated while QRS was notched or slurred in approximately half of the series (Cases II, III, VI and VIII). Four times the principal deflection of the QRS complex was directed downward

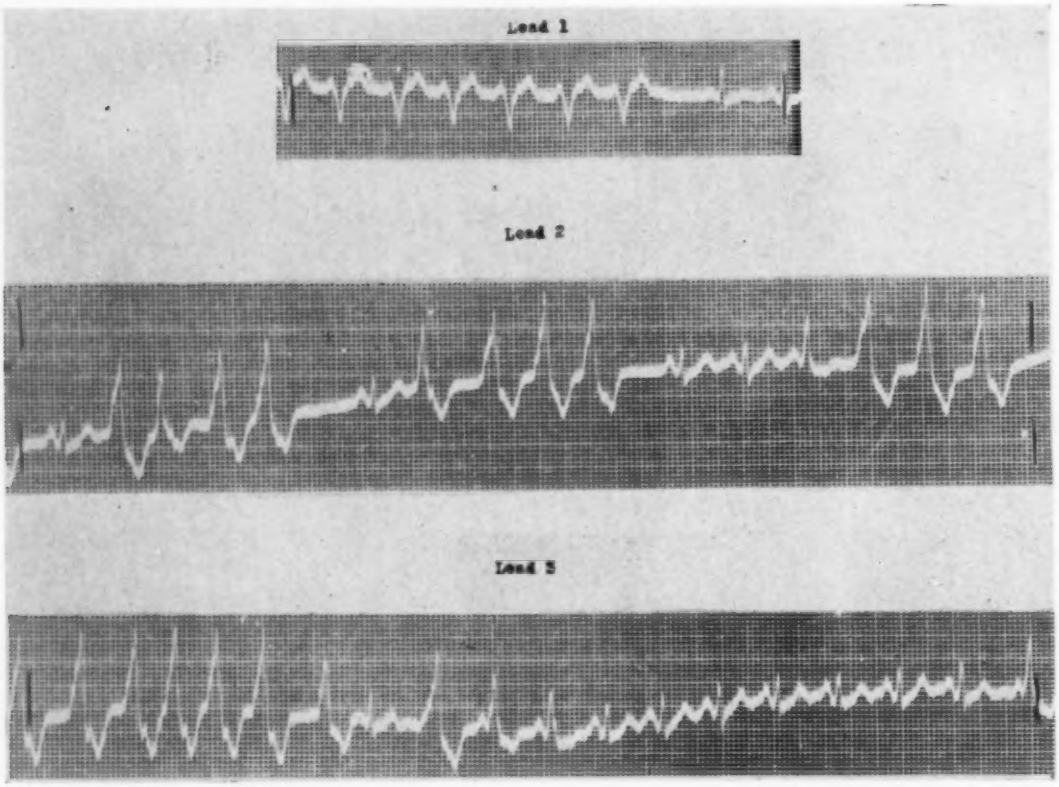


FIG. 12. Tracing taken directly after exercise. The rate of the paroxysms of tachycardia range from 130–188, averaging 146. The rate of the sinus rhythm varies from 120–146, averaging 128. Note the P waves notching the downward limb of the T complex in the ectopic beats in leads 2 and 3 as well as the extremely short PR interval (.07 sec.) in the transition beat occurring in the center of lead 3.

(Cases IV, V, VIII and IX). T Wave changes were common, the complex being diphasic 3 times (Cases II, III and IV), low voltage twice (Cases VII and IX) and inverted twice (Cases V and VIII).*

In summary we may say that minor variations in the electrocardiogram are common in this condition and are probably due to a change in position of the heart attributable to the deformity rather than to myocardial pathology. T wave changes in lead CF_4 were found to be the most constant variation in our series.

One rather interesting case of arrhythmia occurred which was considered worthy of more detailed study (Case II; Fig. 10). This consisted of a bigeminal rhythm which was easily detected by clinical examination. The patient had been told previously that something was wrong with his heart although he had not noted any subjective manifestations, and he was under the impression that he had been placed in 4F draft classification because of a "bad heart." Electrocardiographic tracings taken at rest showed alternating ventricular extrasystoles which had a chief initial deflection directed upward in all leads suggesting a focus of origin in the right ventricle near the conus[15] (Fig. 11 and 14). In this connection it is interesting to point out that von Pohl, Henschen, and Nageli mentioned postmortem findings in cases of funnel chest consisting of grooving of the right ventricle but these evidently were not accompanied by cardiac arrhythmia.[8] The only previous case of disturbed rhythm occurring in association with funnel chest was a patient with paroxysmal auricular fibrillation.[3,8]

* The majority of our patients were young adults.

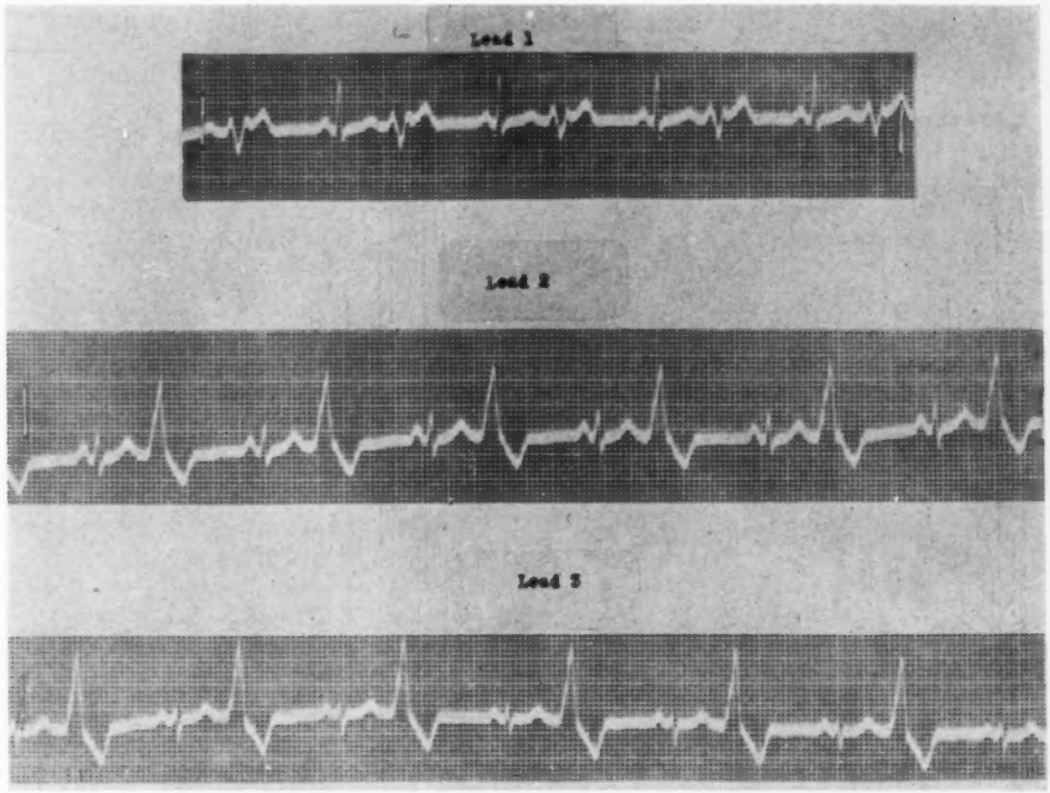


Fig. 13. Tracing taken three minutes after exercise. There has been a complete return to bigeminal rhythm and the form closely resembles that of the control record.

Needless to say, we were well aware of the fact that displacement of the heart will have an effect on the normal electrocardiographic complexes and probably also on the recorded form of premature beats. The concept of the localization of the origin of ventricular extrasystoles as it exists today is based primarily on observation and experimentation involving human and animal hearts. The human observations have the disadvantage of being, in most instances, studies on single cases of diseased hearts,[1,11,12,23] while the animal experiments have the drawback of involving different thoracovisceral relationships which are present in the lower mammalian forms used, for the most part dogs, where there is a difference in the anatomical relation of the ventricles to each other and in the relation of the heart to the chest wall.[15] Most authors agree, however, that to give a chief initial deflection upward in all three leads the stimulus must originate in the right ventricle, near the base, in the region of the pulmonary conus[1,2,4,12,13,15,17,19,21,22,23,24] (Fig. 14). Examinations of the chest roentgenograms of this case show that the above mentioned area is the likely site of bony pressure resulting from the deformity.

Figure 12 shows the effect of exercise (jumping up and down until marked dyspnea was produced) on the rhythm. This tracing shows runs of ventricular paroxysmal tachycardia alternating with periods of normal rhythm. It should be noted that the transition phases in leads 2 and 3 exhibit ectopic beats which differ in form from the other extrasystoles. These represent instances in which the normal and ectopic impulses both enter different parts of the ventricles so nearly at the same time that contraction begins in two places at

once. The resultant beats exhibit a ventricular complex which seems to be a composite of the usual premature beat and of the complex of the normal beat. The runs of extrasystoles occurring after exercise demonstrate a heightened irritability of the ectopic focus which vies with the sino-auricular pacemaker for domination.

The tracing (Fig. 13) was taken three minutes after exercise and represents a

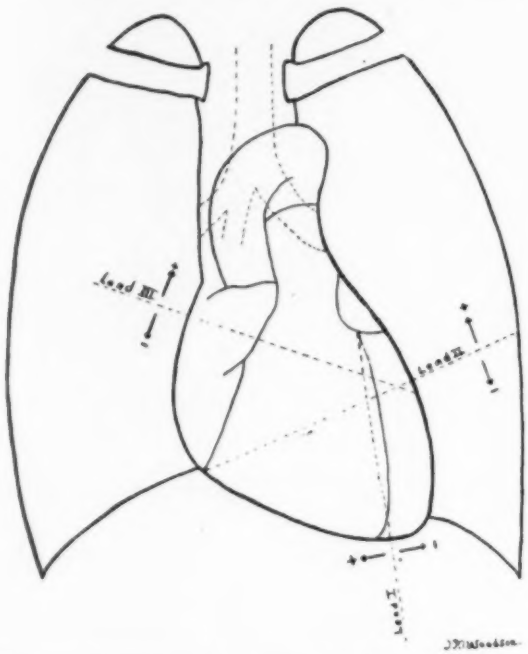


FIG. 14. Diagram of the ventral surface of the heart and great vessels. The dotted lines represent the theoretical planes of demarcation which may aid in predicting the area of origin of an ectopic stimulus from the pattern of the chief initial deflection of the premature beats in the standard leads of the electrocardiogram. It is likely that similar areas may be outlined on the posterior surface of the heart.

complete return to bigeminal rhythm. This reversion was so rapid that it was necessary to exercise the patient between each lead since facilities were not available for taking them simultaneously.

Tracings following the injection of 1/75 grain of atropine showed no change in the character of the bigeminal rhythm.

In the literature funnel chest has been credited with producing various cardiac manifestations such as dyspnea, cough, paroxysms of suffocation,[20] and rapid heart action.[8] Sauerbruch has stated, "... a high grade disturbance of cardiac and pulmonary function may occur with signs of congestion of the peripheral vessels."[3] Recent investigations have tended to question the frequency of these manifestations, however, and we are in agreement with Ediken, Wolforth, and others[8] in the belief that uncomplicated non-traumatic developmental funnel chest usually does not produce symptoms of a serious nature, probably due to the adequacy of the various compensatory mechanisms which have been described.[3,8] When questioned directly, several of our patients said that they had noted dyspnea on exertion and palpitation with exertion or excitement. These complaints were not marked, however, and caused no serious handicap. In this connection Grödel has pointed out that the deformity is often associated with an asthenic habitus which in itself may be associated with dyspnea and fatigue.[8] Moreover, the unusual contact of the heart with the chest wall found in this condition may in itself be productive of a greater or lesser degree of heart consciousness. Chest pain occurred in 4 cases (Cases IV, VII, VIII and IX) but was not characteristic of the type usually associated with organic heart disease. Two patients (Cases VIII and IX) complained of occasional ankle edema but had no other signs or symptoms of cardiac failure. Minor varicosities of the lower limbs were present in one of these (Case VIII). Significant cardiac physical findings were encountered in only one case (Case II). Diastolic clicks and friction rubs previously reported[20] were not found. Blood pressure readings were within normal limits with the exception of a slight systolic elevation in one patient (Case II). In one case (Case VIII) a prominent epigastric pulsation and systolic blow over the abdominal aorta brought up the question of abdominal aneurysm. Slight cyanosis of the nail beds was observed 3 times (Cases III, IV and VIII) but was not considered to be of any

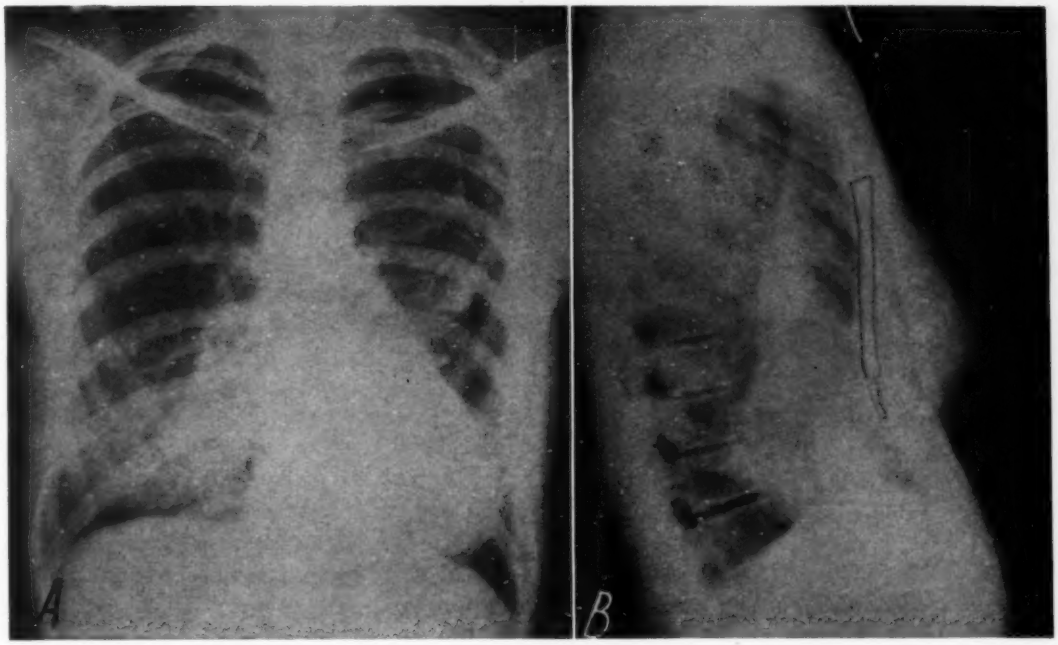


Fig. 15, *A* and *B*. W. F., clerk typist, aged twenty-seven. *Family history:* Not significant. *Past history:* Scarlet fever, migraine, "growing pains" as child, left upper tuberculosis discovered in 1940; pneumothorax initiated at that time and carried to date; has worked full time since 1941. *Symptoms:* Palpitation noted four to five days before each refill when lying on the left side, dyspnea with exertion, occasional nocturia. *Physical findings:* Second pulmonic sound accentuated, grade 2 systolic murmur over the pulmonic area; varies with respiration. Blood pressure, 110/60, distant breath sounds on the left, maximum depression of the lower sternum 3.5 cm. *Electrocardiogram:* Right axis deviation, plus 100 degrees. *Roentgen findings:* There is a pneumothorax with approximately 40 per cent collapse of the left upper lobe. Portions of the left upper lobe are held against the chest wall due to adhesions. The right lung field is clear. The heart is somewhat over to the left side. The lateral roentgenogram shows a moderate funnel chest deformity involving the lower sternum which accounts for the shift of the heart to the left.

significance. Mild hyperesthesia over the fourth and fifth ribs in the left mid-clavicular line was found once (Case VII). No patient exhibited cardiac enlargement although in all but one instance (Case IV) the heart showed the characteristic displacement to the left. Four patients gave a history of the deformity occurring, in a greater or lesser degree, in one or more other members of the family (Cases III, IV, V and VII). The patient in whom bigeminal rhythm was found (Case II) had been classified as unfit for military service because of the deformity and arrhythmia although he suffered no functional limitations and regularly engaged in strenuous exercise without ill effect. Another (Case VII) had been discharged from the Army, after three years of military service including considerable combat, because of poor eyesight.

As shown by previous authors concurring on this subject,[3,8] we found the deformity occurring most often in the lower portion of the sternum. In our series the depth of the depression varied from 3.5 to 7 cm. In the latter case (Case II) the distance from the maximum depression of the sternum to the ventral surface of the spine was 6.5 cm. Verse has recorded a case where this measurement was reduced to 1 cm.[3]

Most cases of developmental funnel chest necessitate no treatment. Surgical elevation of the sternum, however, may have to be considered, particularly in the traumatic group, when progressive cardiac manifestations are present that cannot be attributed to other causes. The case of Sauerbruch

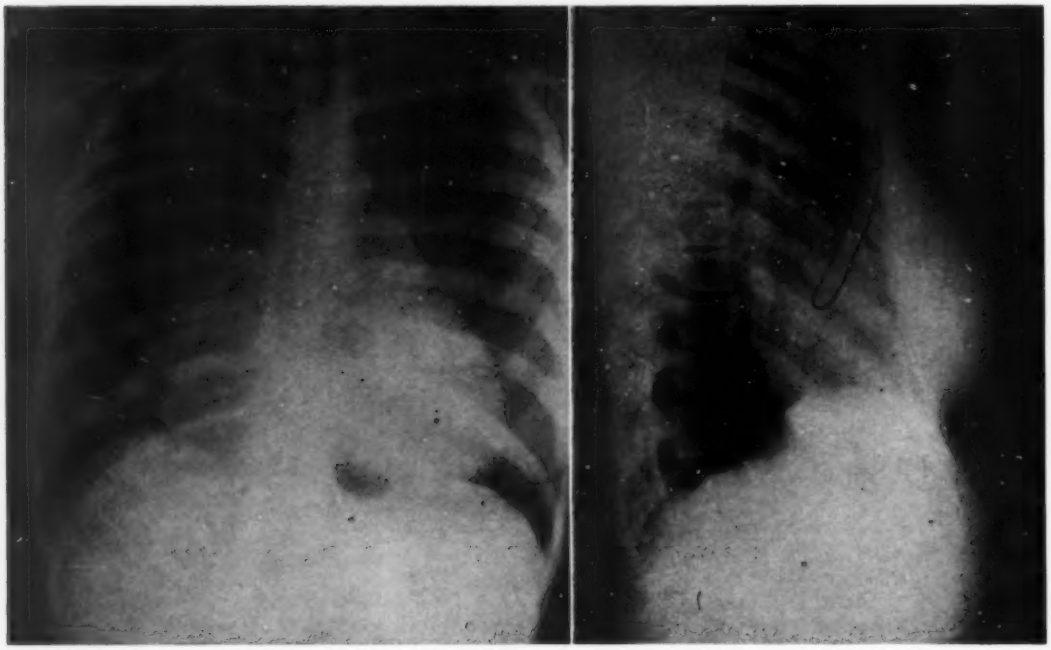


FIG. 16, *A* and *B*. W. F., clerk, aged twenty. *Family history:* Brother has a funnel chest. *Past history:* Scarlet fever, tonsillitis, "kidney trouble." *Symptoms:* Palpitation and dyspnea with exertion, rare fainting spells, occasional fleeting attacks of chest pain located under the upper sternum, relieved by holding breath. *Physical findings:* P2 greater than A2, slurring of M1. Blood pressure, 110/70, maximum depression of the lower sternum 3.5 cm. *Electrocardiogram:* Within normal limits. *Roentgen findings:* There is a moderate funnel chest deformity with shift of the heart into the left chest.

and von Hoffmeister which exhibited paroxysmal auricular fibrillation was relieved by operation.[8] Complaints of a neurocirculatory nature, when present in these patients, are usually not directly attributable to the thoracic deformity and are best treated by a conservative program of graduated exercise. Reassurance may be very important in those cases where the malformation is a cause for excessive concern on the part of the patient.

SUMMARY

1. Nine cases of funnel chest are described, including one very unusual patient with bigeminal rhythm due to regularly recurring right ventricular extrasystoles. This is probably due to an irritable focus in the right ventricle resulting from the constant pressure of the deformed sternum in this region.

2. A characteristic roentgen finding in these cases is displacement of the heart to the left associated with a characteristic appearance of the left cardiac border which can readily be mistaken for an enlarged heart, or a congenital or mitral heart.

3. Although several of the patients noted palpitation and dyspnea, especially after exercise, none of them had any serious cardiac embarrassment. Only one patient had important objective findings.

4. Minor electrocardiographic abnormalities are sometimes present, particularly in the chest leads. These are due to a combination of a shift to the left and a rotation of the heart on its long axis rather than to associated myocardial pathology.

5. Funnel chest is probably quite common. The interpreter of miniature chest films should be aware of the roentgen changes on the posteroanterior roentgenogram to avoid pitfalls in diagnosis.

6. Surgical treatment is rarely indicated. It should not be advocated unless there are clear cut findings of increasing cardiac em-

barrassment not attributable to other conditions.

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ADDENDUM

Since this paper was completed two additional cases of funnel chest have come to our attention (Fig. 15 and 16). One of these (Fig. 15) is especially interesting since there was a left-sided therapeutic pneumothorax present with approximately 40 per cent collapse of the left lung. Despite this fact, the heart showed the characteristic shift to the left.

THE TECHNIQUE OF THE ROENTGENOLOGIC DEMONSTRATION OF PULMONARY INFARCTS*

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CONTRARY to the experience of some others elsewhere in tbis country, at the Massachusetts General Hospital pulmonary infarction has been one of the two most common postoperative complications. It is also one of the frequent medical problems which the internist asks the roentgenologist to study. Since a pulmonary infarct may be the first sign of impending massive fatal embolism, and since today certain preventive measures against fatal embolism are available,[1,2] early recognition of infarcts becomes increasingly important. Valuable technical factors that have been evolved in recent years are of aid in the roentgenologic demonstration of a pulmonary infarct as it becomes apparent. These points merit emphasis since all roentgenologists, those in private practice seeing ambulatory patients as well as those in the hospital encountering ill, bedridden ones, will be called upon at some time to recognize and diagnose pulmonary infarction.

Hampton and Castleman[5] have furnished excellent descriptions, both roentgenologic and pathologic, of pulmonary infarcts in their various stages. Inasmuch as the appearances they depict are revolutionary when compared with former conceptions, the roentgen characteristics will be briefly

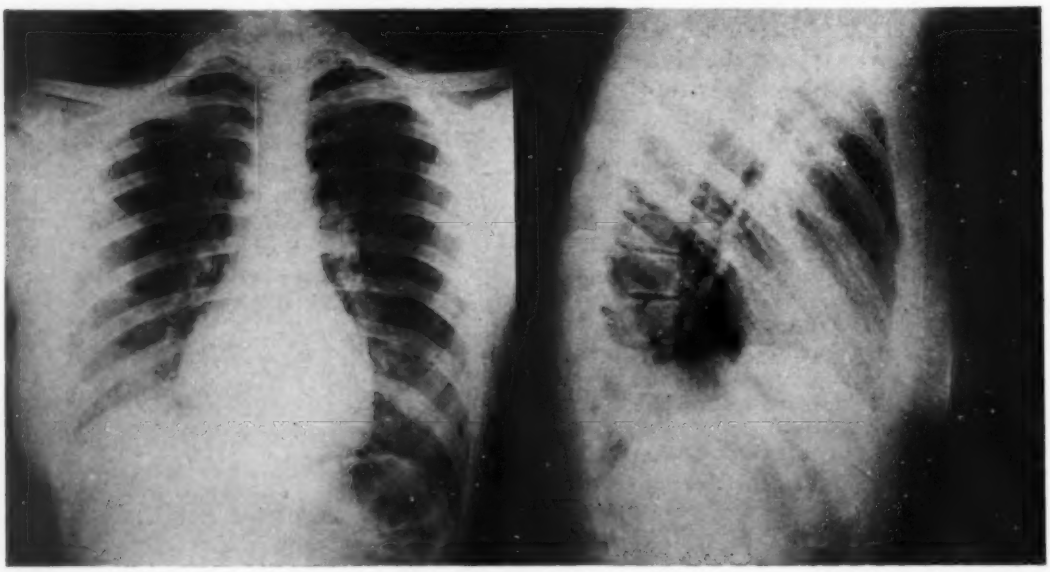

FIG. 1. R.S. (U-425299) Seventeen days after operation for a slipping patella, a nineteen year old girl developed severe pain in the right lower chest and top of right shoulder. The pain was not associated with a chronic cough of long duration. The leg had been in a cast constantly from the time of operation. She was readmitted to the hospital twenty-four hours after the onset of the chest pain. *Roentgen examination:* The anteroposterior view did not demonstrate the lesion. In the lateral view, the shadow seen with the curved anterior margin lying in the posterior right costophrenic angle was thought to represent an infarct.

Bilateral femoral vein ligation was done, and a large thrombus was removed from the right superficial and common femoral veins. Follow-up roentgenograms showed the characteristic appearance of healing infarct.

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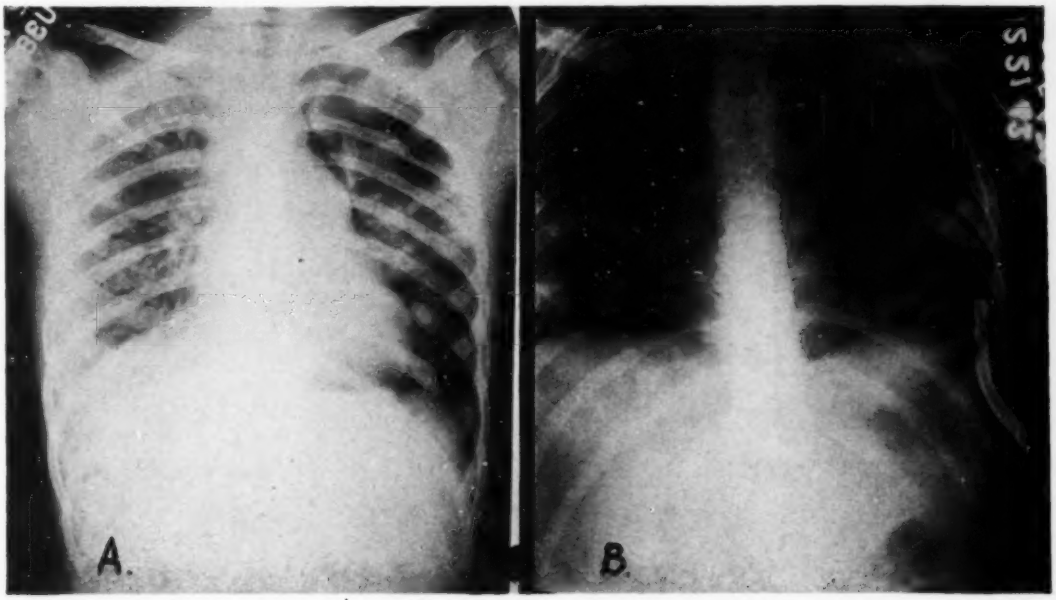

Fig. 2. J.F. (U-385476) A seven year old girl entered the hospital because of marked respiratory difficulty. There was a large fluctuant, reddish-blue swelling over the glabella with edema sufficiently marked to cause the eyelids to be closed. The abscess had developed gradually over a period of ten days. *Roentgen examination:* (*A*) Note pneumothorax on the left and a few mottled areas of increased density scattered through the right lung field. There is also a small amount of fluid in the right pleural cavity. (*B*) Roentgenogram taken ten days later shows a well defined abscess in the left lower lung field and two in the right lower lung field.

Numerous blood cultures were positive for hemolytic *Staphylococcus aureus*. The abscesses healed rapidly, leaving minimal scars.

reviewed. The size of an infarct may vary from a small, thin lesion lying against the pleura to a large lesion occupying the greater portion of a lobe (Fig. 1). The shape of an infarct is dependent upon its location, but one characteristic is constant,—that is, an infarct is always peripheral, with its long dimension lying parallel to the pleura. As a rule its shadow has a curved proximal margin, the convexity being directed toward the pulmonary arterial source of the embolus. An infarct is at first indistinct and gradually it becomes more sharply circumscribed. In a period of time varying from days to months, it assumes a linear shape, and finally it may disappear except for a very thin linear scar.

Approximately 75 per cent of infarcts occur in the lower portions of the lungs, but it must be remembered that they may be found also in the upper lobes. The presence of a shadow in an upper lobe showing the characteristics of an infarct should suggest this diagnosis.

Septic infarcts usually appear as areas of increased density with indistinct margins which gradually become round and sharply defined, and many of them develop a cavity within the center due to bronchial drainage. This type of infarct is a common complication of infections about the face and throat, of osteomyelitis, and of staphylococcal or streptococcal bacteriemia (Fig. 2).

TECHNIQUE OF EXAMINATION

If infarcts are to be suspected and differentiated from other processes within the lung, certain technical procedures must be followed in the roentgenologic examination. Unless the patient's condition forbids, a chest examination should always include roentgenoscopy. The possibility of diagnosing an infarct is of course dependent on securing for study roentgenograms which

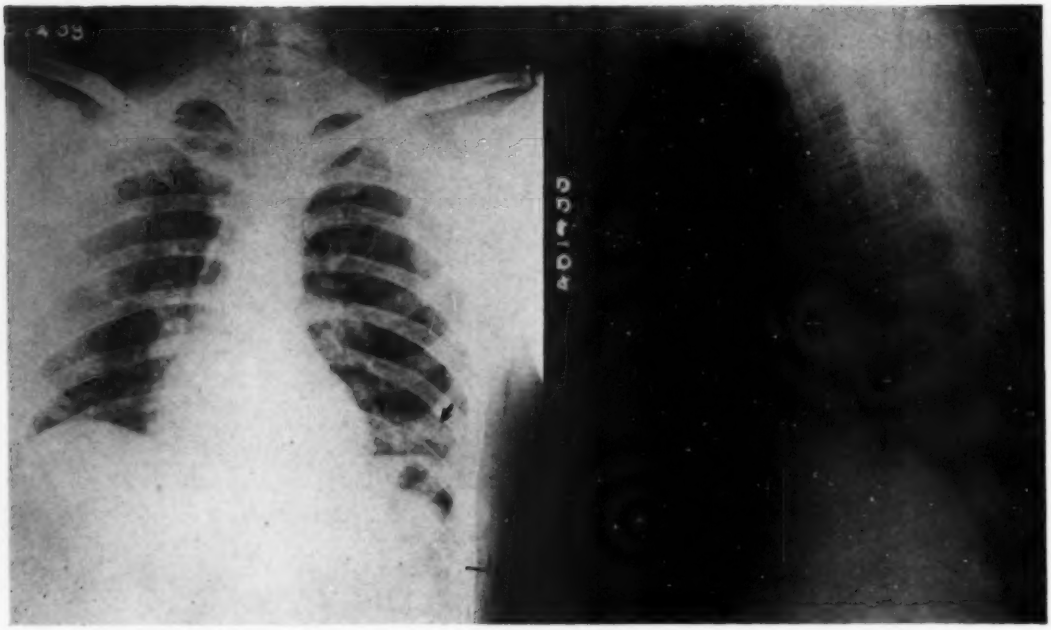

Fig. 3. J.G. (U-461433) Six days following a right lumbar sympathectomy for Buerger's disease, a thirty-five year old man gradually developed pleuritic pain in the left anterior chest. There was tenderness in the left calf which extended into the left thigh. *Roentgen examination:* Note the area of density in the left lower lobe with slightly elevated left diaphragm. This was considered consistent with an infarct. The lesion gradually healed leaving a linear scar.

Bilateral vein ligation was done, and histopathologic examination revealed a few inflammatory cells in the wall of the vessels.

demonstrate it in at least two different projections. During roentgenoscopy it becomes apparent which positions will best serve this purpose. In addition, roentgenoscopy reveals the dynamics of the chest, the position and motion of the diaphragm, and often diagnostic indications that may not be evident on the roentgenogram itself. The leaf of the diaphragm, for instance, which underlies an infarct in the inferior portion of a lower lobe, is usually elevated and splinted (Fig. 3). A small amount of fluid, often the first sign which requires a search for a small area of consolidation, may be detected more easily by roentgenoscopy than on the film proper.

The so-called spot roentgenogram is taken during roentgenoscopy. This type of roentgenogram, which is better known in examination of the gastrointestinal tract, is equally valuable in examination of the chest for demonstration of an infarct or of any lesions in the bronchus or lung (Fig. 4). As in the gastrointestinal study, this type of film should be aimed at the lesion. A spot roentgenogram will often afford a better opportunity for study than will the routine large roentgenogram since positioning of some patients to secure the latter may be difficult.

The roentgenoscopic examination, as a rule, is made at a high kilovoltage, approximately 85 to 95 kv., with a low milliamperage giving a low roentgen output. This protects the patient against overdosage while allowing penetration through the heart and giving better study of the dynamics of the chest than at a lower voltage. The spot roentgenogram is taken at the same kilovoltage with very short exposure, the timing usually being done with the foot rather than by means of a timer.

The completion of the roentgenoscopic examination will leave the roentgenologist

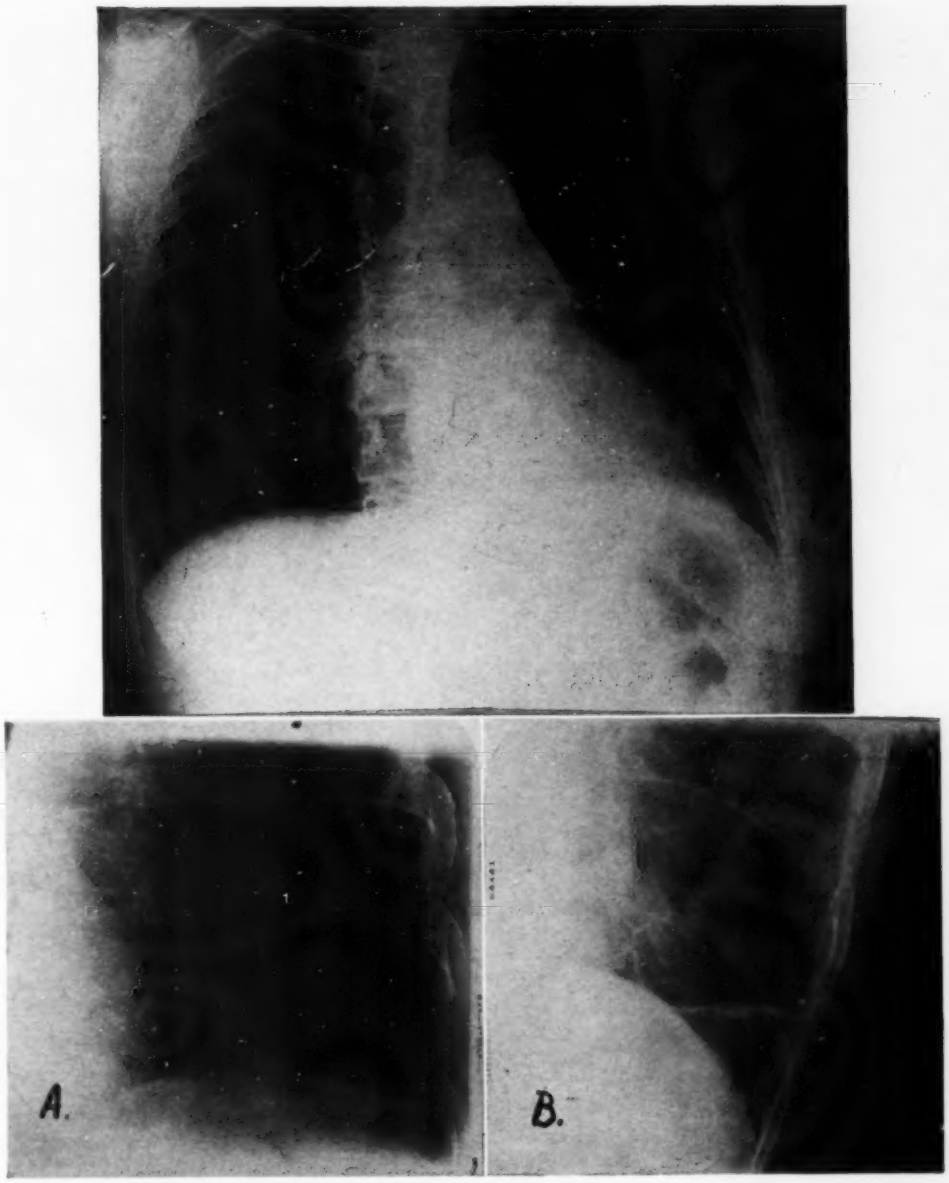

Fig. 4. I.L. (U-432527) Approximately ten days after vaginal hysterectomy, a sixty-three year old woman complained of tenderness in both calves, and ran a slight fever. Several days later she experienced severe pain in the left lower chest. *Roentgen examination:* At this time, the left diaphragm was slightly elevated and showed some limitation in motion. Spot roentgenograms (*A*) taken at the time of the large roentgenogram better demonstrate the lesion lying just above the left diaphragm. (*B*) Spot roentgenogram taken several days later shows the linear shape of the infarct as it healed.

Bilateral vein ligation was done, but no clots were found.

with an understanding of the dynamics of the chest and knowledge of what roentgenograms are necessary to demonstrate the lesion. A posteroanterior roentgenogram is always necessary, and if spot roentgenograms have not been made, a lateral view, or oblique view, is requisite (Fig. 5 and 6). Occasionally a single roentgenogram or stereoscopic views will be sufficient to demonstrate an area of density but not the

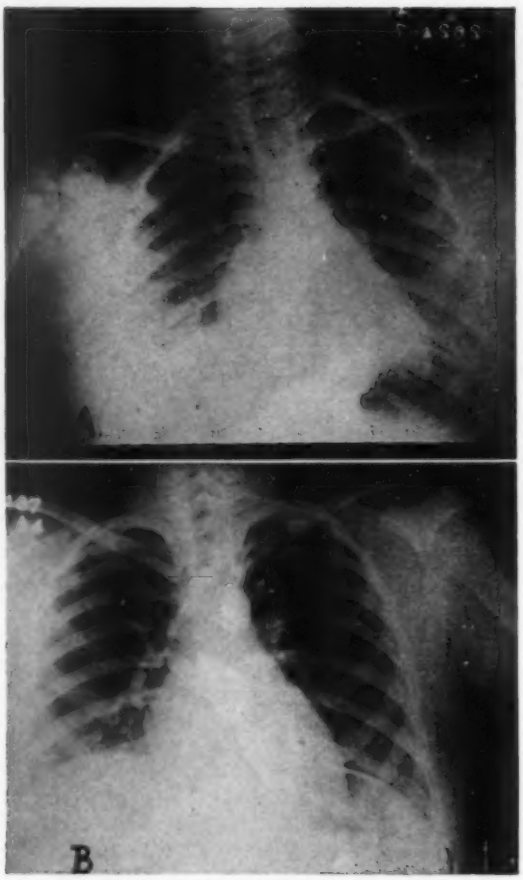

FIG. 5. B.S. (U-262467) A twelve year old boy entered the hospital for removal of a cartilage from the knee. Several days after operation, he complained of pain in the right lower chest and at the same time developed a cough with elevation of temperature, pulse, and respiration. *Roentgen examination:* (*A*) Portable roentgenogram. No definite shadow suggestive of an infarct could be demonstrated. (*B*) Posteroanterior view taken in the Department of Radiology three days later does not demonstrate the lesion.

configuration of an infarct. If the patient is too ill to permit roentgenoscopy, a less complete examination must be made. This is often the case in hospital practice. A single anteroposterior and a lateral roentgenogram, the latter being taken with the patient's affected side directed towards the film, must be made to suffice. It has been our good fortune to have at our disposal a ceiling floor mounted tube stand, beneath which it is possible to push a bed and obtain films of excellent quality at a 6 foot distance and with a short exposure time. This has been of aid in obtaining more accurate diagnosis, and has practically eliminated portable films of the chest which are usually not diagnostic of the small infarct. The patient is subjected to no more manipulation than in examination with the portable apparatus and is kept in the department only as long as it requires to make two exposures.

Bronchography and laminagraphy in our experience have not been of value in demonstrating pulmonary infarcts. Furthermore, in most instances the patient is too ill to stand the manipulation required for such unnecessary procedures.

Certain technical factors have proved of sufficient value to warrant further description. The conventional roentgenograms are all taken with a rotating anode tube which provides a small focal spot, with high milliamperage and short exposure time, thus giving excellent detail. The preservation of the 6 foot distance has been of the greatest value in preventing distortion. A modification of Fuchs' technique,[4] evolved by Oliver E. Merrill, Physicist of the Department of Radiology at this hospital[7] has been, in our opinion, an advance in pulmonary roentgenology. Using the fundamental ideas of Fuchs—a high or better stated optimum kilovoltage for the part, and variation of milliampere-seconds with the size of the patient—a roentgenogram of excellent and more uniform quality has been obtained. This improvement over previous roentgenograms is probably due to the fact that a much greater latitude is possible at a higher than at a lower kilovoltage. Fuchs' technique called for shorter distances than 72 inches, but Merrill's modification makes the 72 inch distance possible, and as has been said, it is the maintenance of this distance which we believe is of the utmost importance in demonstrating pulmonary lesions accurately. Continued research has further improved the quality of the roentgenograms and the present factors used are

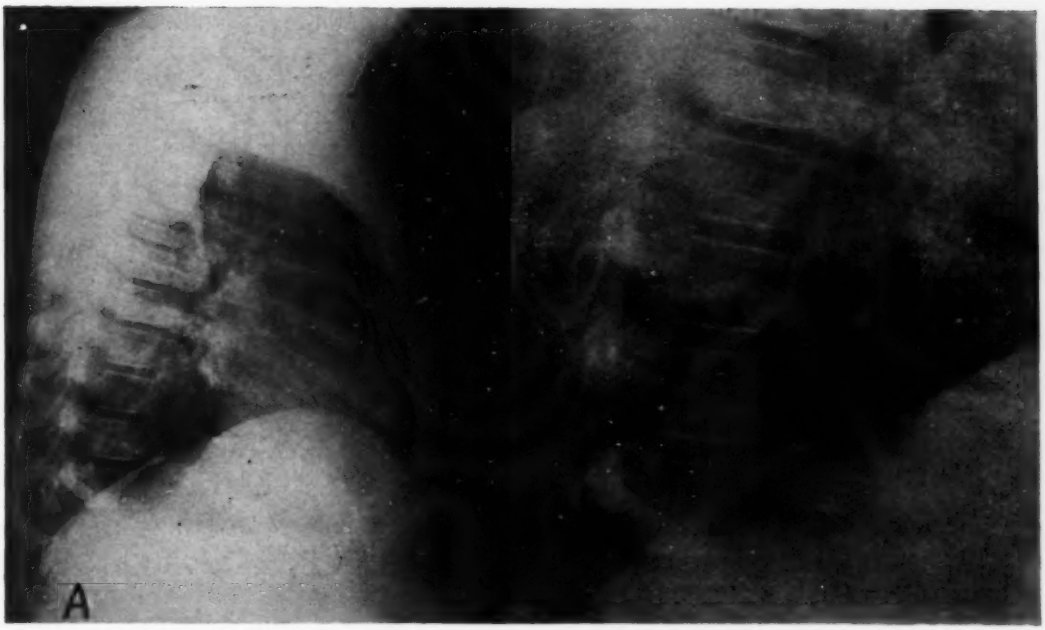

Fig. 6. (Same case as in Fig. 5.) (*A*) Lateral view showing shadow in posterior portion of right costophrenic angle hidden behind the right diaphragm. (*B*) Enlargement of the same area to better demonstrate the shadow of increased density. Note that there is a small amount of fluid posteriorly in the pleural cavity and the posterior few centimeters of the right diaphragm are not visualized.

Although it was believed that the roentgen appearance was consistent with an infarct, the age of the patient made conservative therapy seem advisable. Convalescence was uneventful.

shown in Chart 1.[6]

Roentgenograms taken with this technique have a somewhat steel-gray appearance and show less contrast than those taken with a lower kilovoltage. They are, however, undoubtedly of better diagnostic quality and do not have to be retaken as often as formerly because of technical errors.

DIFFERENTIAL DIAGNOSIS

As in the diagnosis of any lesion, the differential possibilities must be studied and the clinical history considered in making the final decision. Pulmonary infarct, particularly in its healing stage, throws a linear shadow at times almost indistinguishable from that of atelectasis. The work of Fleischner, Hampton and Castleman[3] has clarified to some extent the differential points in these two lesions. Linear areas of atelectasis, which are frequently multiple in the same chest, are of short duration and cast a shadow that may extend to the pleura on each side—it is a long shadow, which shows no peripheral nodularity. The shadow cast by a healing or healed infarct, on the other hand, also extends to the pleural surface but at that point a small nodule is visualized: this shadow disappears slowly, often being seen for some weeks.

Occasionally a pulmonary infarct may reduce the size of a lobe. In such a case, the

CHART I

CHEST EXAMINATION

300 ma. 72 in. distance

Size Chest	Posteroanterior		Oblique		Lateral	
	cm.	sec.	cm.	sec.	cm.	sec.
Small	19	1/60	23	1/20	26	1/15
Average	20–25	1/30	24–30	1/15	27–32	1/10
Large	26–29	1/20	31–33	1/15	33–	1/10
Huge	30–	1/15				
	68 kv.		75 kv.		80 kv.	

differential diagnoses must include other conditions, such as primary carcinoma, that also cause diminution in size. It is rare for an infarct to produce segmental collapse.

Bronchopneumonia or metastatic malignancy may be mistaken for infarction. In pneumonia, however, the shadow seldom becomes sharply circumscribed as in infarction nor does it necessarily extend to a pleural surface. Although metastatic malignancy usually throws a rather sharply circumscribed shadow, its size does not decrease over a period of days but instead tends to increase slowly.

Only the common and most confusing problems in differential diagnosis have been discussed, and the discussion has been of utmost brevity. The point it is desired to emphasize at this time is the importance of technical factors in demonstrating a pulmonary infarct.

SUMMARY

The importance of early recognition by the roentgenologist of pulmonary infarction has become increasingly evident as modern methods for preventing and combatting its sequelae, fatal massive emboli, have been developed. The characteristic roentgenologic appearance (shape, size and location) of a pulmonary infarct from its initial to its healing phase has been reviewed. Certain confusing differential points have been described.

Proper technique of the roentgen examination is the important factor in the roentgenologist's ability to demonstrate a pulmonary infarct. For a satisfactory study, the essential points are roentgenoscopy, spot roentgenograms, and roentgenograms taken in the posteroanterior and lateral projections. Diagnostic accuracy has been greatly improved by the use of the modified optimum kilovoltage technique.

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Vol. 56, No. 6

SIMULATED DEXTROCARDIA ASSOCIATED WITH BRONCHIECTASIS

REPORT OF CASE

By MAJOR S. A. LEADER

MEDICAL CORPS, ARMY OF THE UNITED STATES

DEXTROCARDIA secondary to fibrosis and retraction of the right lung has been well known for many years especially in chronic fibroid tuberculosis, and also in association with bronchiectasis. Primary or congenital dextrocardia with transposition of the viscera accompanied by bronchiectasis, although quite rare, does occur, too, as recorded by Kerley[1] who cites a number of such cases reported by Kartegener and mentions one of his own that came to autopsy. Furthermore, isolated congenital dextrocardia, without the characteristic electrocardiographic changes of the mirror image type of dextrocardia, has been described by White[2] (among others). In view of these facts it is believed the follow-case presents features of sufficient interest to warrant reporting.

REPORT OF CASE

A soldier, white, aged twenty-one, was admitted to this Army General Hospital on October 29, 1944, complaining of shortness of breath on exertion, cough, morning expectoration up to one-half sputum cup daily, occasionally blood streaked, and pain in the right chest.

He gave a history of "rheumatic fever and pericarditis" at the age of eight or ten, which confined him to bed for quite a number of weeks; a year later he developed whooping cough, followed by chronic cough and expectoration ever since. In addition, about once a month during the winter, attacks of sore throat, cough, dyspnea and fever would develop. From the time of his "rheumatic fever," his physical activities were somewhat restricted.

In 1941, after a routine examination for employment, including a chest roentgenogram, he was told he had dextrocardia, but was accepted.

In December, 1942, and again in July, 1943, he was rejected by the Army because of dextrocardia and pericarditis (patient's statement). (Incidentally complete examinations, including electrocardiograms, were made at a civilian hospital after these rejections but no report of the findings was given him.)

In September, 1943, after a paroxysm of coughing, hemoptysis of a cupful of dark red blood occurred. Nevertheless in November, 1943, after a physical examination by an internist, the patient persuaded the latter to give him a letter to the induction board recommending acceptance. Following this he was reexamined by the Army, accepted, and on December 1, 1943, inducted.

Throughout his Army service dyspnea on exertion has been his chief difficulty. After two weeks' basic training, he was hospitalized for observation because of dyspnea but was returned to duty in two weeks. In April, 1944, he again entered the hospital, this time for nasopharyngitis, the symptoms being much like his usual winter attacks. Later that same month he was treated for cellulitis and abscess in the left inguinal region following injury.

Overseas the dyspnea became more marked, particularly in combat, when he was unable to run even in the face of enemy fire. Finally on October 11, 1944 (after the engagement at Brest) he was sent to an aid station and thence to various hospitals until he reached this hospital. At an evacuation hospital a roentgen report of October 12, 1944, stated in part "—mediastinum and cardiac shadows shifted right, right heart border similar to that seen on left. This may be true dextrocardia or only apparent." At a general hospital his chief complaints again were dyspnea and pain in the right chest as if something were squeezing him. His temperature was elevated to 102° F. for one day, then subsided. A roentgenogram of the chest was interpreted as showing an isolated dextrocardia, partly rotated probably congenital; an electrocardiogram was normal. Patient was returned to the United Kingdom for further study and admitted to this hospital on October 29, 1944.

The essential physical findings were: (1) moist medium râles over lower third of right lung, with fine râles at the left base; (2) heart

FIG. 1. Roentgenogram showing dextroposition of heart; shift of mediastinum to right; S-shaped line at right base (displaced primary fissure); pleuropericardial adhesions on right.

on right but greatest intensity of heart sounds in mid-sternal region with equal transmission to either side; (3) no murmurs or thrills were present; (4) cardiac reserve was good. Blood pressure 112/85; heart rate 82; (5) no cyanosis and no clubbing of fingers.

Laboratory Findings. Sputum—half cup of gray sputum daily with pneumococci predominating on culture. Sputum did not form layers on standing and had no perceptible odor. In the examination of the blood, the Kahn reaction was negative; erythrocyte count and sedimentation rate normal; leukocyte count 11,450 and 13,150 with a differential count of 53 per cent polymorphonuclears, 43 per cent lymphocytes, 3 per cent eosinophiles and 1 per cent monocytes. Urinalysis was negative; electrocardiogram, negative (as on examination prior to admission).

Roentgen Findings.

(1) Right-sided heart, mediastinum shifted to right, some narrowing of right hemithorax and slight elevation of right diaphragm.

(2) Emphysema localized to lower half of right lung (middle lobe on lateral view).

(3) Marked downward and backward displacement of right primary fissure, with some twisting—so that the fissure appears as S-shaped linear density extending from extreme tip of right cardiophrenic angle to lower right heart border, with convexity upward, medially and superiorly, and downward, laterally and inferiorly (Fig. 1).

(4) Curved bands of adhesions extending from medial third of right diaphragm to upper right heart border.

(5) Barium-filled gastrointestinal tract normal in position.

(6) Bronchogram showing marked tubular bronchiectasis of right lower lobe affecting the posterior basal branches chiefly, with sac-like collections more laterally at the base. Marked retraction of the right lower lobe as shown by crowding of the bronchi; also slight bronchiectasis of right middle lobe (Fig. 3 and 4); saccular bronchiectasis of left lower lobe medially.

(7) On roentgenoscopy slight rotation produced almost normal cardiac configuration with aortic arch clearly seen on left, and left heart border demonstrated just to left of mid-

FIG. 2. Dorsal and downward displacement; emphysema of right middle lobe.

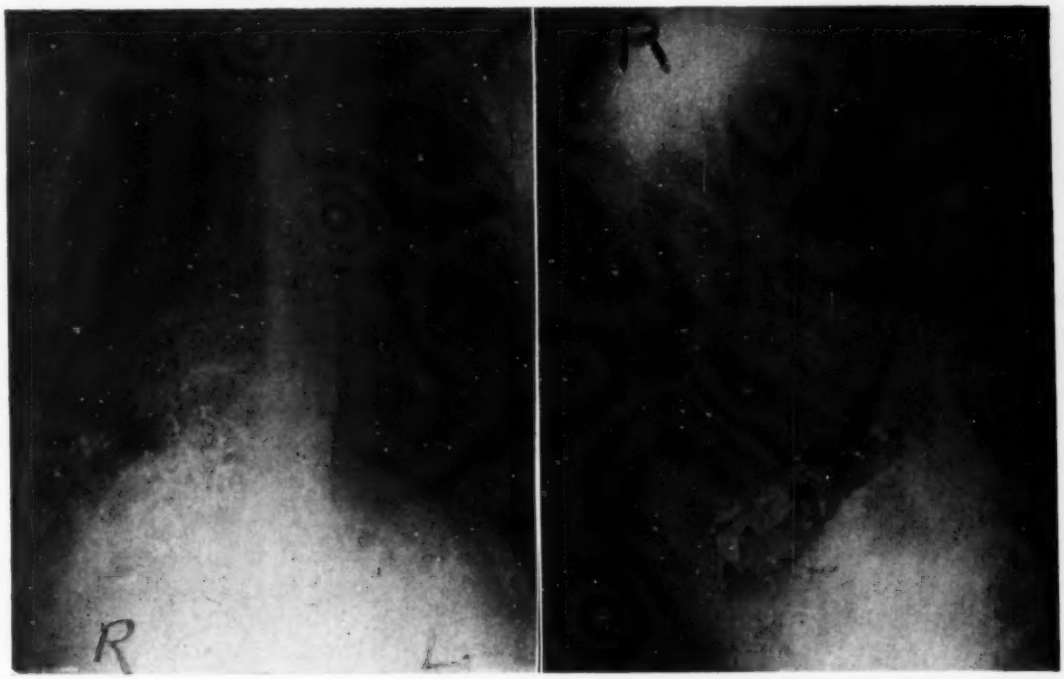

Fig. 3 and 4. Marked bronchiectasis and retraction of right lobe; slight bronchiectasis of right middle lobe.

line. This was confirmed by a roentgenogram made with the same degree of rotation (Fig. 5).

DISCUSSION

These roentgen studies demonstrate clearly that neither situs inversus nor transposition of the cardiac chambers is present. Not so readily disposed of is the possibility of an isolated congenital dextrocardia, suggested by a previous examiner. At this point a brief consideration of the various types of congenital dextrocardia is in order. Two types of the "isolated" form are known; the first without and the second with transposition of the chambers. In the other group, transposition of the chambers occurs with complete or partial situs inversus. (Thus "mirror image" or dextrocardia with transposed chambers may occur with or without transposition of the viscera.) The first form of isolated dextrocardia consists of a right-sided heart, slightly rotated, with the left chambers to the left and anterior, and the right chambers to the right and posterior; and the apex is made up of the right ventricle, or the right side of the common ventricle.[2]

Fig. 5. True relation of cardiac chambers, without transposition demonstrated by slight rotation of patient (pleuropericardial adhesions seen more clearly).

White further states that in this type almost invariably some serious associated congenital anomaly, like a single ventricle, is present. The second or mirror image type of isolated dextrocardia is also said not to occur without other more important congenital cardiovascular defects. White and Kerley both refer to the work of Roesler in 1930 in this connection. Kerley mentions two such cases of his own, with transposition of the chambers but not of the aorta—one of these was found at autopsy to have gross defects of the interventricular and interauricular septum. These had been suspected clinically but were not revealed by roentgen examination. I have also seen a case of isolated "mirror image" dextrocardia in a colored woman (a WAC applicant) with other congenital anomalies (such as ten ribs, and absence of vagina). No other cardiac abnormality was demonstrated and the electrocardiogram was normal.

No statistics on the incidence of electrocardiographic findings in the various congenital dextrocardias just mentioned appear in the references available to me at this time. It has been established that the pathognomonic pattern—an inversion of all the complexes in Lead 1, with Lead 2 resembling the customary Lead 3, and Lead 4 like the usual Lead 2—is found in "mirror image" dextrocardia accompanied by complete situs inversus. Ashman and Hull[4] state that similar patterns occur in "mirror image" dextrocardia with situs inversus, but because of the usual association with other cardiac anomalies the picture is often bizarre. In the group of isolated dextrocardias without transposition of the chambers (classified as one of the main groups by White) it seems evident that any electrocardiographic changes would depend on the associated cardiac abnormalities present.

The present case obviously resembles the first type of isolated dextrocardia, namely without transposition of the chambers. However, it is believed that the rotation and dextroposition of the heart are secondary to the marked retraction of the right lower lobe, shifting of the mediastinum and pleuropericardial adhesions. Further support of this contention is the absence of any other cardiac abnormality. On similar grounds the S-shaped line at the right base is interpreted as a displaced and distorted primary fissure, not ordinarily seen on the posteroanterior view. On the other hand, it may be maintained that this S-shaped line represents the displaced fissure of an inferior retrocardiac accessory lobe—which may be the site of an atelectatic bronchiectasis as demonstrated by Kerley; Kerley cites similar observations by others including Richards. In the present instance, the history and roentgen findings render the explanation of a displaced primary fissure more likely.

Another point deserving mention is the presence of a marked localized emphysema which has been emphasized by Twining[1] as an indication of atelectatic bronchiectasis of the lower lobe. The bronchiectasis of the right lower lobe revealed in this case appears to conform to this type of bronchiectasis as described by Twining. It is interesting to note, however, that in spite of the advanced changes, this is a relatively dry case of bronchiectasis with very little expectoration and the condition could well be simulated by congenital bronchiectasis (which is also dry) with mediastinal shift.

In conclusion, the sequence of events appears to have been pulmonary infection in childhood, followed by bronchiectasis; retraction of right lower lobe, emphysema of the right middle lobe and pleuropericardial adhesions combined to produce dextrocardia with some cardiac rotation. Twining states that probably the majority of atelectatic bronchiectasis in a lower lobe are of the acquired type and the sequence is collapse, infection, bronchiectasis—not bronchiectasis followed by collapse. In this case unfortunately the early history is indefinite and no roentgenograms are available to demonstrate collapse of the right lower lobe which may have been present at the onset.

SUMMARY

(1) A case of apparent dextrocardia is demonstrated to be secondary to atelectatic bronchiectasis and retraction of the right lower lobe with additional factors of emphysema of the right middle lobe and pleuropericardial adhesions contributing to rotation.

(2) Other conditions to be considered in the differential diagnosis are discussed briefly, namely:

(a) Primary dextrocardia with transposition of the viscera associated with bronchiectasis.

(b) Isolated congenital dextrocardia (with bronchiectasis).

(c) Congenital bronchiectasis with mediastinal displacement.

(d) Bronchiectasis of a retrocardiac inferior accessory lobe.

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A CASE OF DIVERTICULOSIS OF THE VERMIFORM APPENDIX ROENTGENOGRAPHICALLY DEMONSTRATED

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DIVERTICULOSIS of the appendix is occasionally seen by the pathologist. However, the diagnosis is rarely made preoperatively. We wish to report the following case in which diverticula were visualized in the appendix during a gastrointestinal examination and later confirmed at surgery.

CASE REPORT

This soldier, white, aged twenty-five, was admitted to the hospital complaining of right lower quadrant abdominal pain, localized at McBurney's point. The patient stated that for the past two years he had had frequent similar attacks of pain in the right lower quadrant. The attacks lasted for one or two weeks, followed by remissions of about two months. The pain was rather severe and well localized. Partial relief was obtained by flexing the thigh on the abdomen. He had been hospitalized once previously for the same complaint but was discharged without surgery. The present attack of pain began about four days previous to admission and became progressively worse. There was no history of constipation, diarrhea, nausea, or vomiting. The review of systems was, otherwise, essentially negative. The family history was irrelevant. Previous history was likewise irrelevant.

Upon admission, the abdomen was soft and flat. There was tenderness in the region of the cecum, extending upward along the usual course of the ascending colon. The tenderness was more marked over McBurney's point. Rectal examination with bimanual palpation revealed a small very tender mass in the cecal region. Admission blood count revealed: erythrocytes, 4,450,000; leukocytes, 8,100; hemoglobin, 90 per cent, with neutrophiles 76 per cent, lymphocytes 20 per cent, monocytes 4 per cent. A routine urinalysis was essentially negative. The blood Kahn reaction was negative.

Roentgen studies of the colon were made by means of a barium enema and a twenty-four hour roentgenogram after ingestion of contrast medium. The colon filled readily on the retrograde study, without evidence of gross deformity or delay. There appeared to be slight irritability of the ascending colon. A small rounded projection defect extended from the appendix near the junction of the mid and distal thirds. This was considered to be a small diverticulum. Another, more questionable, diverticulum was visualized in the same region.

Examination of this patient's colon twenty-four hours after ingestion of an opaque meal showed a very small amount of barium in the appendix, insufficient for complete visualization. However, there was definite subjective tenderness, localized medial to the cecum in a position which was occupied by the appendix when filled. The appendix extended from the cecal tip obliquely upward, the same position shown on examination three months previously. The cecum was not freely movable, and, inasmuch as the appendiceal position was somewhat unusual and, apparently, had been maintained over a period of months, it was assumed that the appendix was fixed in the position described and that the subjective tenderness was attributable to some type of pericecal or periappendiceal pathologic change.

On the basis of the clinical findings, supported by the roentgenological findings, appendectomy was performed on May 3, 1945. The cecum was found to be fixed. The appendix was retrocecal, retroserosal and fixed. The serosal veil of the cecum was dissected from the appendix and the appendix carefully removed.

The pathologic examination of the appendix was reported by the pathologist, as follows:

Gross: The specimen is a curved appendix with a total length of 8 cm. and an average diameter of 0.6 cm. The serosal surface appears slightly congested, but is smooth and glistening. Along the medial concave border, where a small amount of meso-appendix is included, the wall shows three distinct nodular bulgings, of roughly hemispherical shape, but attached to the appendix along a broad base 6 to 8 mm. across.

"Incision is made through the bulgings. The two distal swellings are similar. They disclose a definite lumen filled with soft brown fecal material, and continuous with the main lumen of the appendix, which is also so filled. The wall is markedly thinner around these diverticula than in the main portion of the appendix, and the muscularis can be seen fading off to nothing. The most proximal swelling, on incision, shows no communication with the central lumen. At this level, the lumen is only a narrow slit in the main appendix, while the diverticulum is filled with soft brown fecal material. The wall of the diverticulum is considerably thicker than in the other distal examples.

"*Microscopic:* Sections through the appendix and diverticula show a dumb-bell shaped lumen filled with fecal material. Leukocytes are not present. The mucosa is everywhere intact but relatively narrow. In the main part of the appendix there is a moderate amount of lymphoid tissue in the submucosa. The muscularis, of average width, has a horse-shoe shape. At the beginning of the diverticulum, the muscle fibers spread out thinly and extend downward around the diverticulum for a very short distance. In the diverticulum proper, a band of muscle fibers is present immediately beneath the mucosa, forming a distinct muscularis mucosae continuous with the muscularis mucosae of the appendix proper, but much more prominent. External to this, there is a thin layer of fibrous and adipose tissue, containing numerous lymphocytes. It is probable that a few delicate muscle fibers persist within this fibrous investment.

"*Diagnosis:* Diverticula of appendix, with very slight chronic inflammatory reaction."

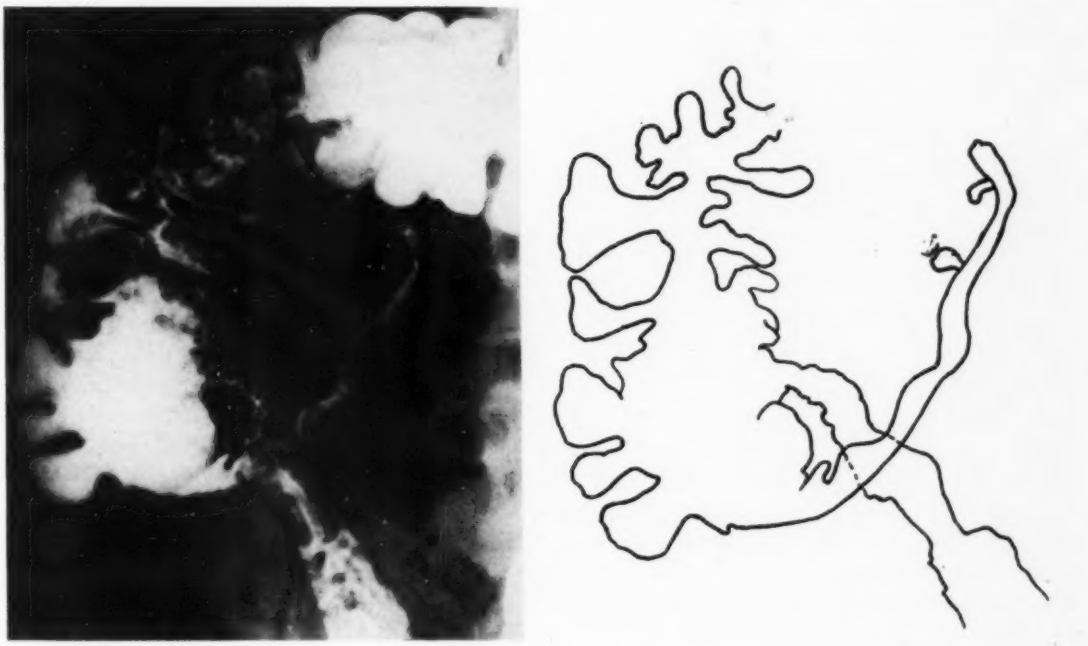

Fig. 1. The terminal ileum, cecum with attached appendix and ascending colon are shown as visualized on the post-evacuation roentgenograms, made at time of colon examination by barium enema. Two small diverticula may be seen projecting from the medial aspect of the appendix as designated in diagram.

The patient made an uneventful recovery and was discharged to duty on May 21, 1945.

Follow-up examination, July 21, 1945, was made. The patient volunteered the information that he felt fine and had had no more abdominal complaints since he was discharged from the hospital. He further stated that, prior to his surgery, any walking induced an attack similar to those described in the admission history, but now he can walk long distances without abdominal discomfort.

DISCUSSION

The incidence of diverticula of the ap-

FIG. 2. Surgical specimen—appendix. Three diverticula are shown projecting into the meso-appendix.

pendix has been variously estimated at less than 0.5 per cent, to as high as 5 in 264 appendices (1.89 per cent) reported by Stout.[1] In this hospital, the pathologist found 4 in 1,850 appendices (0.22 per cent).

The demonstration, preoperatively, by means of roentgen examination, is apparently much more rare than the above statistics indicate. Feldman[2] states that he did not demonstrate any in 20,000 gastrointestinal examinations. He quotes Spriggs and Marxner as having demonstrated appendiceal diverticula in 6 cases and Edwards, in 2 cases. Kadrnka and Sarasin[3] reported a roentgen study of 2 cases in which diverticula were found incidentally. In neither case were there symptoms referable to the right lower quadrant. We have found 1 case in a series of 3,932 gastrointestinal examinations.

Pathologically, all diverticula of the appendix are considered to be of the "acquired" type, there being two groups: (1) those that occur through defects for the passing of the vessels supplying the submucosa and mucosa in the muscular coats, and (2) those which occur through defects in the muscularis due to acute inflammatory processes.[1] It is not our purpose to discuss the pathological morphology, except to point out that our case, apparently, belongs in group (1), since evidence of chronic inflammatory change in the appendix was minimal.

FIG. 3. True cross section of appendix on plane through middle diverticulum. Appendiceal lumen above; meso-appendix below.

The authors are indebted to Dr. Lester S. King, now pathologist at Illinois Masonic Hospital, Chicago, Illinois, for the pathologic material used in this case report.

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Vol. 56, No. 6

A CYLINDRICAL RADIUM APPLICATOR FOR THE TREATMENT OF SURFACE VAGINAL LESIONS*

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THE design of a radium applicator intended for vaginal neoplasms, like most radiotherapeutic instruments, should meet the following requirements:

(a) accurate placement and immobilization of the radiating sources.
(b) uniformity of dosage over the lesions.
(c) adaptability to varying size, extent and location of lesions in organs of different anatomical dimensions.
(d) adequate protection of healthy tissue.
(e) minimum exposure to the persons involved in its preparation and use.

These desiderata, except (c) and (e) can be satisfied by suitable moulages in which radium tubes are inserted to fit the individual patient.[1]

The applicator herein proposed, while not ideal, meets all conditions to a large degree. It must be regarded, however, as a versatile one only in the treatment of superficial lesions limited to the lower three-quarters of the vagina. It consists essentially of two parts, a transparent sheath and a lead core, both illustrated in Figure 1, and shown in cross section in Figure 2.

The sheath consists of a lucite cylindrical tube closed at one end. On its external surface there is permanently scratched a cross-sectional pattern consisting of labelled straight lines parallel to the axis of the shell and of numbered circles perpendicular to them.

The core consists of a lead cylinder on the surface of which a suitable number of longitudinal slots have been milled to accommodate the radium. It is provided with a handle and with a thin transparent cover of plastic permanently attached to it, as shown in Figure 1. This complete core slides into the outer case through a pin-and-slot arrangement. The core envelope is marked similarly to the outer sheath and both sets of markings coincide when the core is fully inserted. It is well to mention here that the straight lines are parallel to the axis of the radium tubes and that the circular scratches are spaced at intervals equal to the length of the radium tubes.

The procedure for determining the portion and the area of the applicator to be filled is as follows. The outer sheath is properly placed in the vagina and, with the aid of a headlight and a laryngeal mirror, the contour of the lesion is inspected through the wall of the plastic tube (Fig. 3). The area covered by the tumor is determined by noting its relationship to the longitudinal and circular marks on the plastic sheath. Since the radium tubes will lie under the longitudinal lines between the circular ones, care is taken to note what lines will extend to or slightly beyond the edges of the cancer. The inner core is then filled by the technician as directed by the

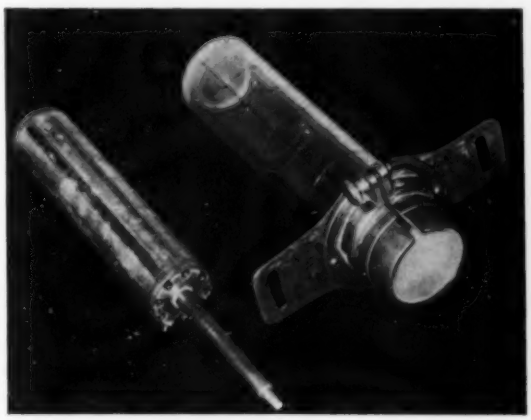

FIG. 1. Transparent sheath and cylindrical lead core shown filled with radium tubes.

* Read at the Twenty-eighth Annual Meeting, American Radium Society, San Francisco, Calif., June 28–29, 1946.

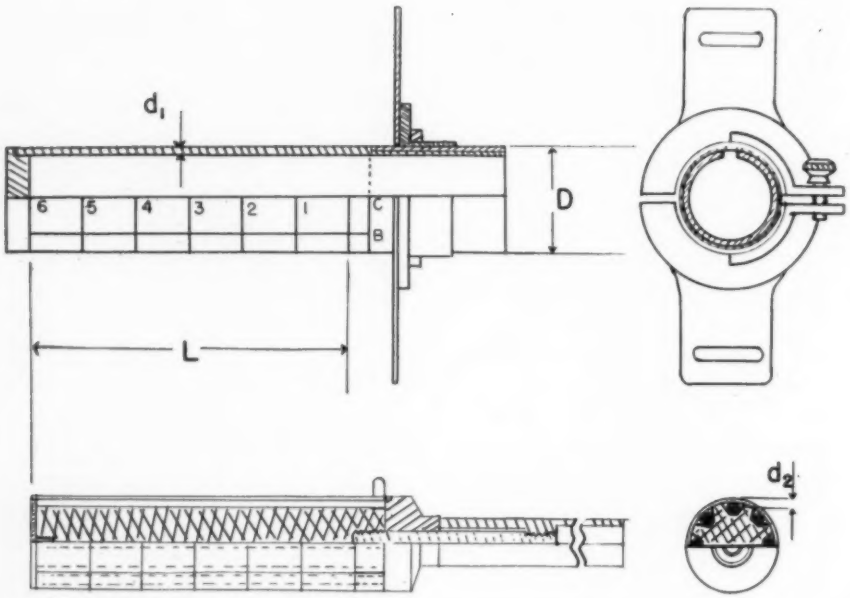

FIG. 2. Cross-section drawing. $L = 9.0$ cm., $d_1 = 0.3$ cm., $d_2 = 0.35$ cm. Large applicator: 8 longitudinal slots, $D = 3.0$ cm. Medium applicator: 6 longitudinal slots, $D = 2.5$ cm.

examiner. The tubes are positioned with the use of dummies in the unoccupied spaces in the core. Since they are discernible from the outside, their position is checked visually by inserting the core into the sheath before delivery to the therapist.

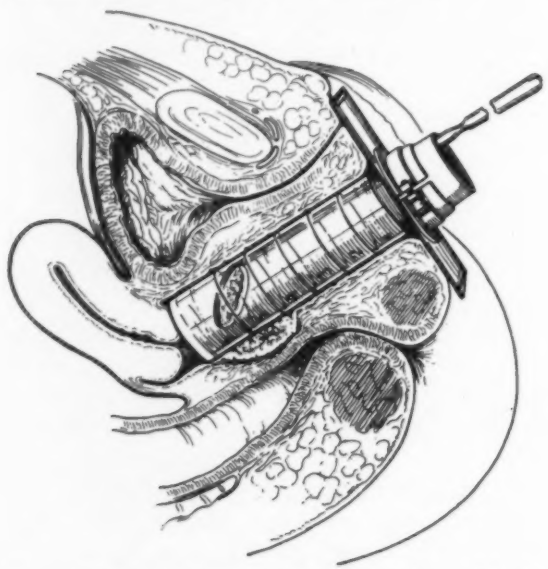

FIG. 3. Cross section of the female pelvis with the outer sheath of the applicator in position, showing how the tumor is visualized with a laryngeal mirror through the transparent lucite wall.

When treatment is given, the outer shell alone is first carefully fitted in the vagina, as was done when determining the area covered by the tumor. The position is checked again with the headlight and the mirror. The shell is then immobilized by means of a split collar provided with slotted flanges which can be secured with a T belt. When this operation is completed, the loaded core can be placed in it (Fig. 4) with

FIG. 4. Core loaded with radium tubes being inserted into the vaginal sheath.

complete assurance as to the accuracy and permanence of position of the radium and with a minimum of exposure to the therapist.

Three sizes of the applicator have been made and the dimensions of the two larger ones are indicated in the legend of Figure 2. The large one, 3 cm. in diameter, accommodates eight slots, and the medium one, 2.5 cm. in diameter, six slots of radium tubes. A smaller type, not shown, is 2 cm. in outer diameter and accommodates three slots in a semicircle; it has been used only in cases of very extensive stenosis. No other sizes have been required by our Gynecological Service.

It can be readily appreciated that the arrangement of parallel tubes on a cylindrical surface can lend itself to innumerable sizes and locations of lesions. Dosage calculations, however, cannot be easily adapted from published data on account of the presence of the lead core and of the impossibility of placing tubes at right angles to each other.

The relationship between exposure (mg-hr.) and dosage in space (r) was determined experimentally for a single tube by means of roentgenographic films and a bakelite phantom. The latter, shown in Figure 5, consisted of concentric tubes of bakelite spaced to accommodate the films and to average unit density over the whole mass. The entire applicator was placed in the center, the first film immediately on its surface, the others at proper distances up to 3 cm. beyond. The usual precautions necessary for this type of work, namely simultaneous processing of all films and calibration under standard conditions, were observed. Thus roentgens were computed on the basis of 8.4 r per mg-hr. at 1 cm. from a point source filtered by 0.5 mm. of platinum. Control standardization of film (density vs. roentgens) was carried out with a radium standard at 7 cm. from the film, the latter covered by $\frac{1}{8}$ inch bakelite on both sides. Densities were measured with a Marshall transmission densitometer. Exposure times in the phantom were adjusted to yield readings on the most convenient portion of the *I-D* curve of the film.

Once the spatial distribution from a tube was satisfactorily determined and found essentially independent of the position of the radium source in the slot, the study was extended to several combinations of tubes by mapping the surface of the sheath rolled out on a plane. Under this condition the lettered slots and numbered rings of the sheath will be represented by intersecting lines. The experimentally determined dose from one tube to a given position can then be read at these intersections and added to the doses from other tubes situated elsewhere. This calculation was carried out at the surface of the applicator and 0.5 cm. beyond.

FIG. 5. Bakelite phantom used to determine distribution of radiation by use of films.

To cover the field systematically, the calculations were first made for an area one tube long and two slots wide; then for the

TABLE I

MEDIUM APPLICATOR

A	CM²	S	D	A	CM²	S	D	A	CM²	S	D
I	0.5	50	165	II	1.5	8	240	I:I	2.5	105	300
I I	1.0	85	240	II II	2.0	130	330	I:I I:I	4.0	165	410
I I I	1.5	120	300	II :: II	4.0	175	430	I:I ::: I:I	8.0	220	485
I I I I	2.5	155	370	II II II II	5.5	215	480	III I:I I:I III	11.5	245	590
I I I I I	4.0	190	425	II II II II II	7.5	265	570	III I:I I:I I:I III	16.0	335	725

A	CM²	S	D	A	CM²	S	D	A	CM²	S	D
IIII	4.0	120	327	IIIII	5.5	145	375	IIIIII	7.5	155	395
I::I I::I	6.0	190	460	IIIII IIIII	7.5	200	470	IIIIII IIIIII	11.5	265	520
IIII I::I IIII	11.5	255	550	IIIII I:::I IIIII	15.0	295	516	IIIIII :::::: IIIIII	23.0	315	665
IIII I::I I::I IIII	17.0	320	685	IIIII I:::I I:::I IIIII	22.5	360	750	IIIIII :::::: :::::: IIIIII	34.0	375	790
IIII I::I I::I I::I IIII	23.0	380	770	IIIII I:::I ::::: I:::I IIIII	31.0	430	825	IIIIII :::::: IIIIII :::::: IIIIII	45.0	425	890

A: Arrangement of tubes in rows and columns.
CM²: Surface area.
S: Milligram-hours per 1,000 r at surface.
D: Milligram-hours per 1,000 r at 0.5 cm. depth.

same height three slots wide, etc., until a ring one tube high was completed: the calculations were then repeated for an area two tubes in length and two slots in width, and so on until a length of five tubes or 7.5 cm. was filled.

At the beginning, for practical reasons, it was decided to use in these applicators tubes of strengths in the ratio of 2:1 (in our case 50 and 25 or 25 and 12.5 mg.) and by a cut-and-try method the most satisfactory arrangement of tubes for the uniform dos-

TABLE II

LARGE APPLICATOR

A	CM²	S	D	A	CM²	S	D	A	CM²	S	D	A	CM²	S	D	A	CM²	S	D
I	0.5	85	210	II	1.5	120	270	I:I	2.5	160	330	I::I	3.5	195	400	I:::I	5.0	230	475
I				II				I:I				I::I				I:::I			
I	1.0	145	280	II	3.0	195	350	I:I	6.0	250	435	I::I	9.0	300	510	I:::I	11.0	340	590
I				II				I:I				I::I				I:::I			
I	1.5	190	355	::	5.0	265	475	:::	9.0	310	525	::::	14.0	385	630	:::::	18.0	430	715
I				II				I:I				I::I				I:::I			
I				II				I:I				IIII				IIIII			
I				::				:::				I::I				I:::I			
I	2.5	235	445	::	6.0	320	590	:::	15.0	400	650	I::I	19.0	465	780	I:::I	24.0	535	890
I				II				I:I				IIII				IIIII			
I				II				III				IIII				IIIII			
I				II				I:I				I::I				I:::I			
I	4.0	280	520	II	8.0	360	635	I:I	18.0	460	755	I::I	25.0	560	900	I:::I	33.0	625	1010
I				II				I:I				I::I				I:::I			
I				II				III				IIII				IIIII			

A	CM²	S	D	A	CM²	S	D	A	CM²	S	D
I::::I	6.5	255	525	I:I:I:I	7.5	275	540	IIIIIIII	10.0	290	575
I::::I				I:I:I:I				IIIIIIII			
I::::I	14.0	380	660	I:I:I:I	16.0	390	680	IIIIIIII	20.0	400	695
IIIIII				IIIIIII				IIIIIIII			
I::::I	23.0	490	805	I::::::I	27.0	525	890	::::::::	36.0	560	910
IIIIII				IIIIIII				IIIIIIII			
II::II				IIIIIII				IIIIIIII			
I::::I				I::::::I				::::::::			
I::::I	30.0	605	1000	I::::::I	36.0	590	1030	::::::::	48.0	665	1110
II::II				IIIIIII				IIIIIIII			
IIIIII				IIIIIII				IIIIIIII			
I::::I				I::::::I				::::::::			
I::::I	40.0	710	1150	::::::::	48.0	770	1250	::::::::	65.0	800	1335
I::::I				I::::::I				::::::::			
IIIIII				IIIIIII				IIIIIIII			

A: Arrangement of tubes in rows and columns.
CM²: Surface area.
S: Milligram-hours per 1,000 r at surface.
D: Milligram-hours per 1,000 r at 0.5 cm. depth.

age distribution over a given area was devised.

The final results are shown in Table I for the medium applicator and in Table II for the large one. In the column marked *A* the full lines and the dotted lines represent tubes of double and single strength respectively. The column marked CM² gives the size of the lesion conveniently covered by this radium configuration.

In the next columns the exposures in milligram-hours are given for a dose of 1,000 r at the skin surface and at 0.5 cm. depth respectively.* Both dosages are uniform, in most arrangements, to within ± 5 per cent throughout the area limited by the mid-points of the corner tubes, and to within 10 per cent in the rest. In practice, after the tumor outline has been determined, the radium is placed to cover the

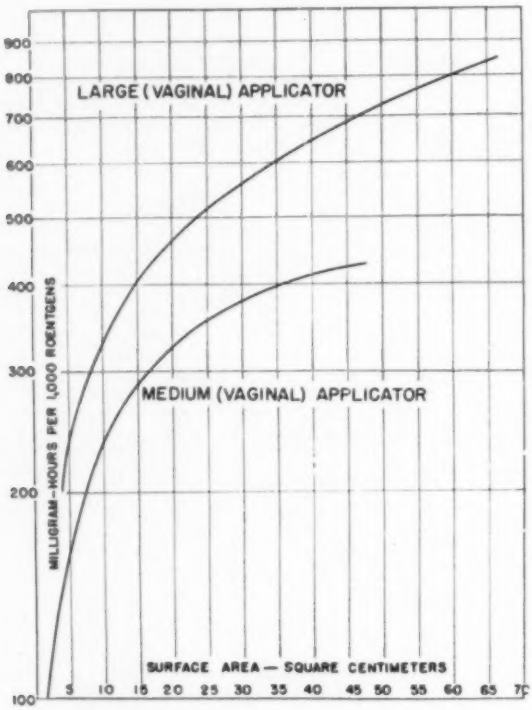

FIG. 6. Curves giving exposure in milligram-hours to produce a dose of 1,000 r at the surface of areas indicated.

nearest rectangle which will extend just beyond the boundaries of the tumor. If the lesion extends beyond the borders of a rectangle but not throughout the length of one side, one or more tubes should be used to cover it. In this case, as well as in the one of very irregularly shaped lesions, a fairly uniform treatment can be given by measuring the area encompassed by the tubes and by determining the exposure (in mg-hr.) necessary to deliver a given dose in roentgens with the aid of the curves shown in Figure 6. Thus, if a lesion 35 cm.^{2} is to be given 5,000 roentgens with the medium applicator, the appropriate curve of Figure 6 shows that an exposure of 400 mg-hr. corresponds to a dose of 1,000 r. Hence a dose of 5,000 r will be obtained with an exposure of $\frac{5000}{1000} \times 400 = 2{,}000$ mg-hr. Figure 6 has been drawn on the basis of the rectangular arrangements shown in the tables. Its validity for irregularly shaped lesions is approximate and requires placement of stronger sources on the periphery and weaker sources in the interior.

To date there are 30 patients whose subsequent records are available to assess the early results obtained with this applicator. The most numerous group and the one in which the most satisfactory results have been obtained are 11 cases of primary carcinoma of the vagina. Of these, 9 are alive and 2 are dead. Of the 11 cases, 7 showed prompt regression of the local lesion with no evidence of recurrence to date. In 2 of these 7 distant metastases in nodes appeared subsequently. In general, the failure to control the primary tumor in 4 cases seemed to be due to the wide extent of the lesion at the time of treatment.

Ten cases of advanced carcinoma of the cervix with extension down the wall of the vagina have been treated by the applicator. Of these, 9 are dead and 1 has been treated within the last six months and the outcome remains in doubt. In 3 cases the vaginal cancer regressed completely after the treatment. In other cases the result of the treatment was hard to evaluate since the patients rapidly deteriorated from extension of their primary tumor into the parametria or from other complications of advanced cancer of the cervix. Little information can be obtained from this group since the prognosis in such cases is almost always very poor. At least the applicator adds one

* The reader is reminded that the dose is expressed in roentgens and that the exposure is expressed in milligram-hours. The dose depends not only on the exposure but also on the geometrical configuration and on the filtration. Therefore the relationship between them must, in general, be established in each instance.

more logical method of treatment of vaginal extensions in this type of advanced cancer.

The remaining 9 cases consist of 4 who had vaginal metastases from cancer of the corpus, in 2 of which the local lesions were controlled, 2 cases of cancer of the urethra, 1 of which has been cured to date, and a case each of endometrial sarcoma metastatic to the anterior vaginal wall, carcinoma of the rectum metastatic to the vagina, and Hodgkin's disease involving the anterior vaginal wall and urethra. All 3 of the latter cases are still alive; the ultimate result of the vaginal treatment remains still in doubt.

For the therapist who desires to avail himself of our experience in using this form of radium applicator, a word should be added as to the dosage used. This has ranged from 3,200 r to 11,300 r on the surface with a single application. The tendency of late has been to give repeated doses a week or two weeks apart, the largest dose being 5,000 r on each of two occasions to a lesion 25 cm.^{2} in area.

So far only 2 instances of radium necrosis have followed the use of the present applicator. The first resulted in a rectovaginal fistula in a patient who received 4,000 r on each of two occasions over a lesion 24 cm.^{2}. She has remained free of evidence of persistent cancer since then. The second was an area of superficial necrosis in the anterior vaginal fornix in the patient with Hodgkin's disease and is probably attributable to the fact that she received simultaneous roentgen therapy since the dose with the applicator was only 3,600 r to a 5 cm.^{2} area on the surface of the vagina.

In treating primary cancer of the vagina, doses have varied from 3,500 r to 11,300 r on the surface and from 1,600 r to 5,000 r at a depth of 0.5 cm. Four cases were treated with two applications of 3,800 r, 4,000 r, 4,200 r, and 5,000 r each. The average single dose was 4,800 r on the surface, the dose of 11,300 r being excluded from the computation. In the latter case the tumor was not controlled and the patient died eight months later. In this small series no correlation can be made between size of dose and cure of disease. The size of the tumor, instead, seems to be the main factor since tumors of 10 cm.^{2} or under seem to have been controlled with one exception, whereas those of larger size have continued to grow.

In cancer of the cervix with vaginal metastases, surface doses of 3,200 to 11,000 r have been given, the average being 5,700 r. The dose at 0.5 cm. depth averaged 3,100 r.

In the remaining patients, surface doses of 3,600 to 8,600 r were given. The 8,600 r was delivered to an anterior vaginal wall metastasis from a primary carcinoma of the corpus a few square centimeters in area. In this case the reaction was sharp but has healed without necrosis and the tumor metastases seems to have regressed completely.

Our experience is too limited to draw conclusions as to any relationship between dose and effect on various types of tumors. All that can be said is that surface doses up to 8,000 or 9,000 r, or possibly higher, in small lesions seem to be tolerated without producing permanent radiation necrosis.

SUMMARY

An applicator has been designed with the purpose of treating nearly all sizes and shapes of superficial vaginal lesions, and of providing adequate protection for the rest of the vagina.

A cylindrical sheath of clear lucite is first placed in the vagina and the outline of the area to be treated is determined by looking at it through the wall of the sheath with a laryngeal mirror. A lead core containing slots along the length of its surface is filled with radium tubes to cover this area. In actual treatment, the lucite sheath is accurately reinserted with the help of the head light and mirror and properly secured. The loaded lead core is then locked in place.

Tables have been constructed to show the practical arrangement of tubes necessary for uniform irradiation of rectangular lesions and the relationship between milli-

gram-hour exposure and tissue roentgens both at the surface of, and at 5 mm. depth from the vaginal wall.

Thirty cases have been treated. These include eleven primary carcinomas of the vagina, ten cancers of the cervix with vaginal extension, four carcinomas of the corpus with vaginal metastases, two carcinomas of the urethra, a case of Hodgkin's disease of the urethra, an endometrial sarcoma metastatic to the vagina, and a carcinoma of the rectum with a vaginal metastasis.

Primary carcinoma of the vagina responded well, seven of the eleven cases showing complete regression of the primary tumor. Cancer of the corpus uteri with vaginal metastases also are suitable for treatment. Cancers of the cervix with vaginal extension have not responded very favorably, probably because of the extent of the cancer in areas other than the vagina.

Surface doses used have varied from 3,200 to 11,300 roentgens at the surface of the vagina. Doses of 8,000 to 9,000 r in small lesions seem to be tolerated without permanent radiation necrosis.

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IONIZATION MEASUREMENTS WITH BONE CHAMBERS AND THEIR APPLICATION TO RADIATION THERAPY*

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THE ordinary depth dose measurements and isodose charts give information concerning the relative expected effect of irradiation on soft tissue. They do not take into consideration the bony structure which modifies the effect in two important aspects. The bones absorb more radiation than the soft tissue and therefore appreciably reduce the depth dose in the beam passing through a thick layer of bone. The bone itself is, on the other hand, exposed to more secondary electron radiation (beta rays) than the soft tissue. Particularly important is the fact that the secondary radiation varies a great deal with the wavelength of the roentgen rays.

A rather thorough study of the problems involved has been published by Juris.[8] The investigation was supported by the Curie Radium Institute in Paris. We started our study before this publication was available and believe our data are of value both for corroboration and for extension of the knowledge in this field.

The effect of roentgen rays on tissues is due to the electron radiation (beta rays) produced and the ionization in a thimble air chamber is due mainly to the secondary electron radiation coming from the wall of the chamber. It therefore seems evident that an ionization chamber made of bone should give an indication of the effectiveness of the radiation on bone. The secondary electrons from the wall consist of Compton electrons, produced during scattering, and photo-electrons. The number of the former is independent of the wall material but the number of photo-electrons varies with the atomic number of the elements in the wall. It increases rapidly with the atomic number for radiation of long wavelength, but only slightly for radiation of short wavelength, such as gamma rays. The path of the secondary electrons is short and amounts to about 0.1 mm. in tissue for 200 kilovolt electrons.

The following equipment was used for the measurements. A bone cylinder of 10.6 mm. inside diameter, 24 mm. length and 0.5 mm. wall thickness was made from a dry human femur, and a cap of the same material, outside diameter and thickness, was attached to one end. The other end slipped over a polystyrene insulating rod to an extent of 6 mm. so that an ionization chamber was formed with a volume of 1.6 cc. The inside electrode consisted of a 0.8 mm. diameter aluminum wire. The other part of the attachment was made similar to the removable Victoreen ionization chamber with nearly the same capacity. The bone ionization chamber thus could be used interchangeably with the ordinary "air wall" chamber in a Victoreen roentgenmeter which was used for the measurements.

One bone cylinder with a wall thickness of 2 mm. was made to fit over the ionization chamber and a second one with a wall thickness of 2.3 mm. could be slipped over the first one. Measurements could thus be taken with chambers of 0.5, 2.5 and 4.8 mm. thick bone.

A chamber similar in size and shape was made of lucite with a wall thickness of 0.5 mm. Measurements with this chamber gave

* From the Department of Radiology and Physical Therapy of the University of Minnesota and The University Hospitals, Minneapolis, Minn. Aided by the research funds of the Graduate School of the University of Minnesota. Read at the annual meeting of the Minnesota Radiological Society, May 26, 1945.

TABLE I

COPPER (CU) FILTER ALWAYS FOLLOWED BY 1 MM. ALUMINUM (AL)
TIN (SN) FILTER ALWAYS FOLLOWED BY 0.25 MM. CU AND 1 MM. AL

100 kv. (peak)		140 kv. (peak)		220 kv. (peak)		400 kv. (peak)	
Filter	Half-Value Layer	Filter	Half-Value Layer	Filter	Half-Value Layer	Filter	Half-Value Layer
(mm.)	(mm. Cu)	(mm.)	(mm. Cu)	(mm.)	(mm. Cu)	(mm.)	(mm. Cu)
0	0.05	0	0.09	0	0.42	0	1.42
1.0 Al	0.09	1.0 Al	0.12	0.125 Cu	0.70	0.5 Cu	2.3
0.125 Cu	0.15	2.0 Al	0.17	0.25 Cu	0.97	1.0 Cu	3.2
0.25 Cu	0.25	0.125 Cu	0.27	0.5 Cu	1.34	2.0 Cu (or 0.44 Sn)	3.8
0.5 Cu	0.35	0.25 Cu	0.42	0.75 Cu	1.63	3.0 Cu	4.2
0.75 Cu	0.42	0.5 Cu	0.58	1.0 Cu	1.90	4.0 Cu (or 0.88 Sn)	4.4
1.0 Cu	0.47	0.75 Cu	0.69	1.25 Cu	2.12	5.0 Cu	4.7
1.25 Cu	0.52	1.0 Cu	0.77	1.5 Cu	2.21	6.0 Cu (or 1.32 Sn)	4.8
1.5 Cu	0.55	1.25 Cu	0.86	2.0 Cu	2.44	8.0 Cu	4.9
2.0 Cu	0.60	1.5 Cu	0.93	3.0 Cu	2.65	10.0 Cu	4.9
		2.0 Cu	1.05	4.0 Cu	2.8	12.0 Cu	5.0
		2.5 Cu	1.15	4.75 Cu	2.9	1.32 Sn + 15.5 Cu	5.2

results which were directly proportional to the Victoreen chamber within 1 per cent over the whole range of roentgen rays used when the 0.8 mm. diameter aluminum electrode was used.

Another bone chamber with 1.1 mm. wall thickness was used for some of the measurements. The measurements with this chamber did not deviate more than 5 per cent from the 0.5 mm. bone chamber.

It should be noted that the small surface of the bone ionization chamber through which the aluminum electrode enters, consists of polystyrene instead of bone. Some measurements were, however, taken with a bone cap covering this surface but as the values did not then change more than 10 per cent this precaution seemed unnecessary. Errors due to the fact that the inside electrode consisted of aluminum rather than bone and to the rather large size of the chamber should be of the same order of magnitude. Such errors, perhaps amounting to 10 per cent, would be of little importance and would not change the conclusions obtained.

Juris found the dry bone was a satisfactory conductor for the wall of the chamber and that it was unnecessary to coat the inside of the conducting surface. This is very fortunate as even a very thin layer of graphite stops a high percentage of the secondary electrons, as will be shown in a curve plotted from measurements with a graphite lined bone chamber. The coating could not be applied with uniform thickness but it could be determined that it was less than 0.1 mm.

TABLE II

EQUIVALENT WAVE LENGTH SCALE

Half-Value Layer	Wave-length Å	Half-Value Layer	Wave-length Å
mm. Cu		mm. Cu	
0.05	0.45	0.500	0.20
0.082	0.40	1.065	0.15
0.15	0.35	2.32	0.10
0.198	0.30	3.18	0.075
0.282	0.25	4.0	0.063

Juris also used fresh undried bone and found that the ionization in such a chamber differed very little from one made of dry bone.

Measurements were first taken in air and later at different depths in a specially constructed water phantom.[9]

Table I gives the peak voltages and the filters used and the corresponding half-value layers in copper as determined by absorption curves. Table II shows the half-value layer for certain wavelengths of homogeneous radiation.[7]

In order to obtain information about the amount of radiation absorbed by bone, measurements were taken with both the bone chamber and the lucite chamber, first uncovered and then with the two bone cylinders over the chambers. The values obtained with one of the chambers covered with bone, divided by the values measured with the uncovered chamber gives the per cent transmission through 4.3 mm. bone. The same transmission figures were found with both chambers and are given for the different half value layers in Table IIIa. They are also plotted in the lower portion of Charts I and II. The points fall close to the curve which represents values calculated from data on bone absorption[1,7] for different wavelengths of homogeneous rays (Table IIIb). It is important to note that in therapy as much as two-thirds of the rays are absorbed by 0.5 cm. bone when 100 kv. (peak) unfiltered radiation is used. With 140 kv. (peak) and 0.25 mm. copper filter

Table IIIa

4.3 mm. bone added to bone or air wall chambers

100 kv. (peak) Half-Value Layer	Per Cent	140 kv. (peak) Half-Value Layer	Per Cent
mm. Cu		mm. Cu	
0.05	33.0	0.09	42
0.09	42.0	0.12	51
0.25	73.0	0.42	82
0.35	80.0	0.69	87
0.42	85.5	1.15	91
220 kv. (peak)		400 kv. (peak)	
0.42	76.5	1.4	87
0.70	81.0	3.8	95
0.97	86.5		
1.34	89.5		
1.63	94.0		
2.44	95.0		

Table IIIb

Transmission 4.3 mm. bone

Half-Value Layer	Per Cent	Half-Value Layer	Per Cent
mm. Cu		mm. Cu	
3.93	96.3	0.694	90.0
3.35	95.7	0.502	87.1
2.39	95.3	0.389	84.8
1.84	94.6	0.246	78.8
1.41	93.4	0.068	43.3
1.14	92.5	0.042	27.0

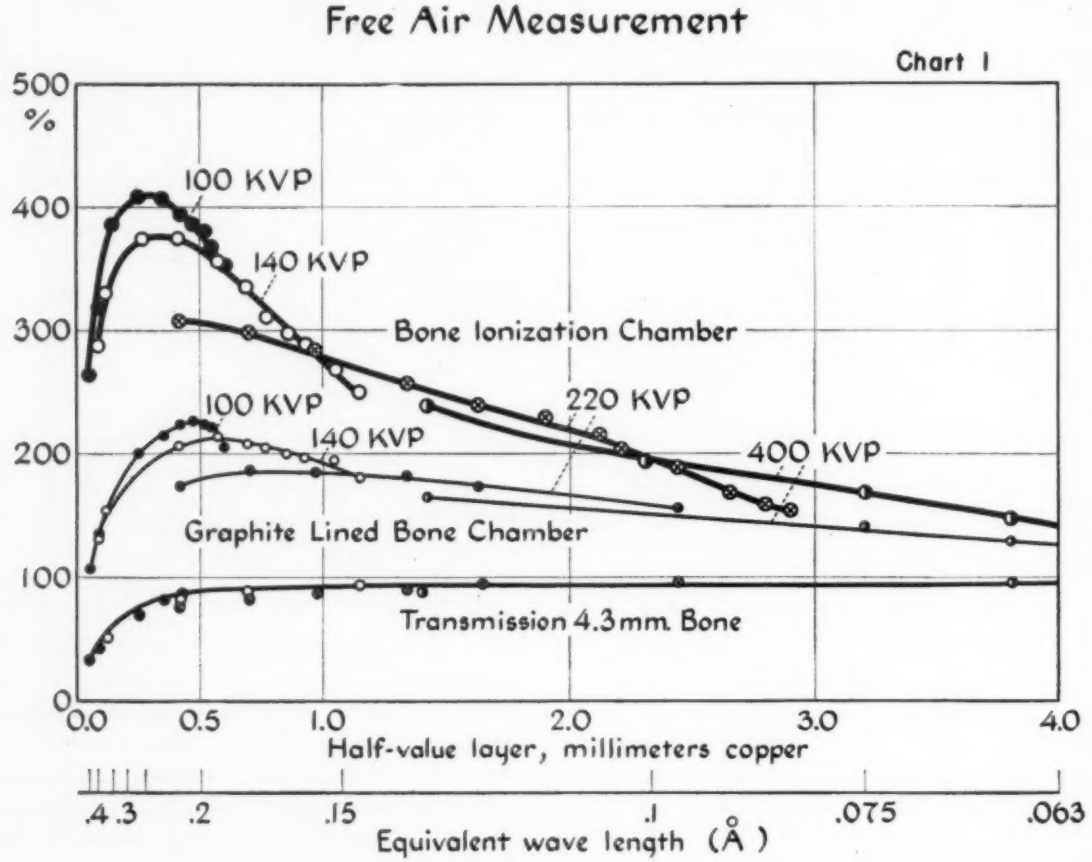
Table IV

Free air measurement—bone ionization chamber (0.5 mm. wall)

100 kv. (peak) Half-Value Layer	%Bone /Lucite	140 kv. (peak) Half-Value Layer	%Bone /Lucite
mm. Cu		mm. Cu	
0.05	263	0.09	285
0.09	318	0.12	330
0.15	385	0.27	373
0.25	408	0.42	374
0.35	406	0.58	355
0.42	395	0.69	333
0.47	386	0.77	310
0.52	380	0.86	297
0.55	366	0.93	288
0.60	352	1.05	267
220 kv. (peak)		400 kv. (peak)	
0.42	307	1.42	238
0.70	297	2.3	192
0.97	283	3.2	168
1.34	256	3.8	147
1.63	238	4.2	138
1.90	226	4.4	134
2.12	213	4.7	129
2.21	201	4.8	128
2.44	188	4.9	124
2.65	168	4.9	122
2.8	159	5.0	121
2.9	153	5.2	115

about 20 per cent is absorbed by 0.5 cm. bone and the absorption is as high as 5 per cent even when 400 kv. (peak) and 2 mm. copper filter is used.

Of importance for the measurements with the bone chamber is the fact that 0.5 mm. absorbs a high percentage of the soft radiation and it amounts to more than 25 per cent for half-value layer 0.05 mm. copper. clearly in Chart 1 where the obtained figures are plotted against the half-value layers. The homogeneous wavelengths of the corresponding half-value layers are marked on a second scale. The curve for 100 kv. (peak) shows a maximum at a half-value layer of 0.3 mm. copper of over 400 per cent. The ionization in bone thus can be expected to be more than four times as great as in soft

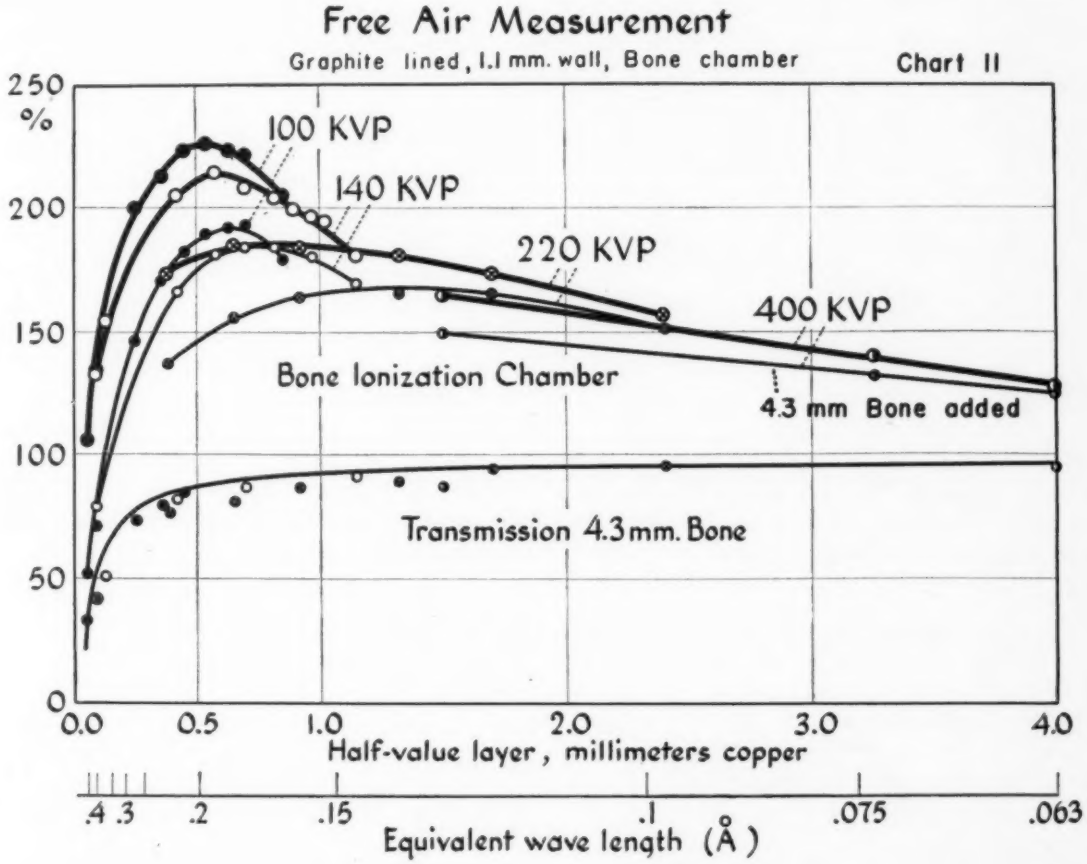


In order to obtain a clear picture of the intense ionization produced in bone, the readings of the r-meter obtained with the bone ionization chamber are expressed in percentage of the readings obtained with the lucite chamber. These are the figures which will be found in the following tables and charts.

Table IV gives the figures obtained for different voltages and different half-value layers when the uncoated 0.5 mm. wall bone chamber was used in air. The trend is shown tissue for this type of radiation. The maximum can be expected to be even higher if a thinner wall is used, as 0.5 mm. bone absorbs a considerable amount of the radiation. The decline of the curve with softer radiation is undoubtedly due to the increased absorption and no maximum point would be expected if correction for absorption were applied. As mentioned before, more than 25 per cent correction should be applied for half-value layer 0.05 mm. copper.

The graph shows, as was to be expected, that the ionization in the bone chamber gradually declines towards the values for the "air wall" chamber with increasing half-value layer and reaches a value of 147 per cent for half-value layer 3.8 mm. copper (400 kv. (peak) and 2 mm. copper filter). Through the courtesy of Dr. Edward Schons of St. Paul, a few measurements were taken with his 1,000 kilovolt machine, with a half-value layer of 9.5 mm. copper (filter: 1 mm. lead + 0.5 mm. copper). A value of 108 per cent was obtained. Measurement with gamma rays from radium filtered with 0.5 mm. platinum gave a result of 104 per cent. This is identical with that given by Smereker.[11]

One of the bone chambers with a wall-thickness of 1.1 mm. had the inside surface coated with a thin layer of graphite. Unfortunately the coating was somewhat uneven and its thickness could therefore not be determined, though it can be stated that it did not exceed 0.1 mm. at any place. The values of these measurements shown in Table v and plotted in Chart II, indicate to what extent the secondary beta rays are absorbed by this layer of graphite. (Compare with Table IV and Chart I.) The ionization contributed by the beta rays from the bone has been reduced to about one-half by this thin layer of graphite.



The size of the ionization chamber may conceivably influence the result as some of the secondary beta rays will not reach the center of the ionization chamber. A smaller bone chamber may therefore give higher values. In order to obtain some information regarding this factor, bone and lucite chambers were made with about half the diameter of the original chambers (5.5 mm. as compared to 10.6 mm.). The curves in

TABLE V

FREE AIR MEASUREMENT—GRAPHITE LINED (INSIDE) BONE CHAMBER (1.1 MM. WALL)

100 kv. (peak) Half-Value Layer	% Bone /Lucite	140 kv. (peak) Half-Value Layer	% Bone /Lucite
mm. Cu		mm. Cu	
0.05	106	0.09	132
0.09	134	0.12	154
0.25	200	0.42	205
0.35	213	0.58	214
0.42	223	0.69	208
0.47	226	0.77	204
0.52	223	0.86	199
0.55	221	0.93	196
0.60	204	1.05	194
		1.15	108
220 kv. (peak)		400 kv. (peak)	
0.42	173	1.42	164
0.70	185	3.2	140
0.97	184	3.8	128
1.34	181		
1.63	173		
2.44	156		

Chart v shows the results with the two sizes of chambers. It can be seen that no appreciable difference occurs for well filtered radiation with a half-value layer greater than 0.5 mm. copper (equivalent wavelength of 0.2 Å). For 220 kv. (peak) radiation, with inherent filtration only (0.2 mm. copper), a 4.4 per cent increase is obtained with the small chamber. The main difference is found for very soft radiation and the increase for the small chamber amounts to 23 per cent for 100 kv. (peak) unfiltered radiation with a half-value layer of 0.05 mm. copper.

The measurements with the small chamber thus indicate that the trend of increased secondary bone radiation towards lower half-value layers is even more pronounced than shown in Chart 1. The difference is, however, of secondary importance and does not affect the main conclusions. The maximum value for the ionization in bone is about 500 per cent.

It is well known that the absorption and scattering of tissues will change the spectral composition of the rays so that the half-value layer increases for very soft rays and decreases for hard rays with increasing depth. Measurements were therefore taken at different depths in the water phantom for the different types of radiation which

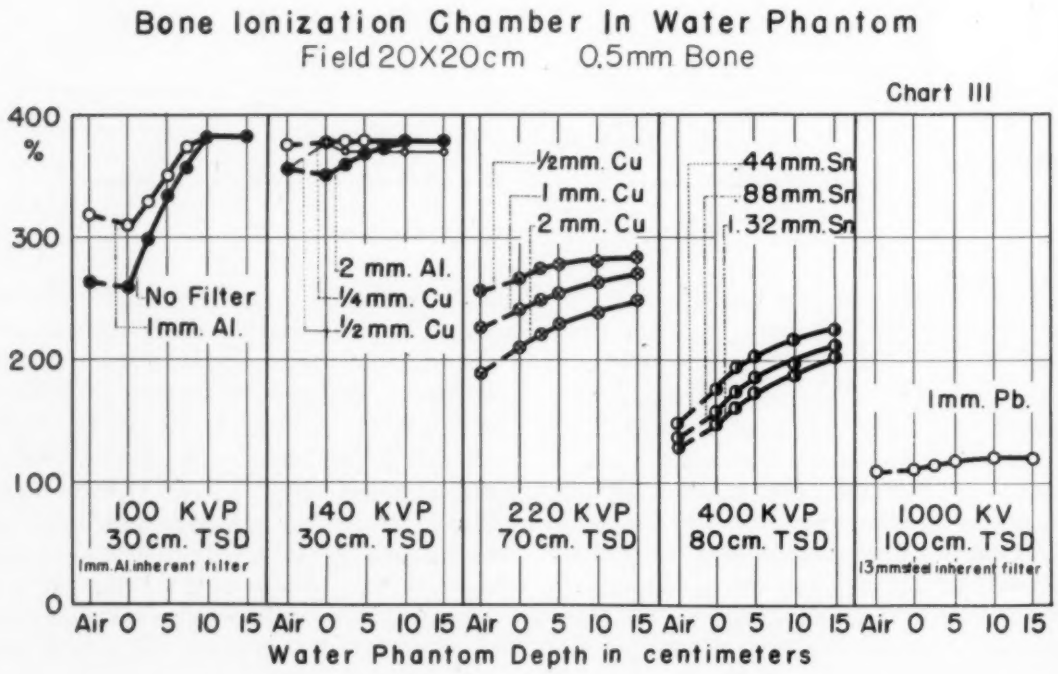


TABLE VI

BONE IONIZATION CHAMBER IN WATER PHANTOM—FIELD 20×20 CM. %BONE/LUCITE

Depth	100 kv. (peak)	30 cm. target skin distance	Depth	140 kv. (peak)	30 cm. target skin distance	
cm.	(1 mm. Al inherent filter) No filter	1 mm. Al	cm.	2 mm. Al	0.25 mm. Cu +1 mm. Al	0.5 mm. Cu +1 mm. Al
Air	263	318	Air	355	374	355
Surface	260	310	Surface	350	377	379
2.5	298	329	2.5	358	378	370
5.0	333	350	5.0	369	378	370
7.5	356	373	7.5	371	378	370
10.0	382	382	10.0	378	378	370
15.0	382	382	15.0	378	378	370

Depth	220 kv. (peak)	70 cm. target skin distance	
cm.	0.5 mm. Cu +1 mm. Al	1 mm. Cu +1 mm. Al	2 mm. Cu +1 mm. Al or 0.44 Sn)
Air	256	226	188
Surface	265	240	210
2.5	273	248	220
5.0	277	253	228
10.0	280	263	238
15.0	283	270	248

Depth	400 kv. (peak)	80 cm. target skin distance		Depth	1000 kv.	100 cm. target skin distance
cm.	0.44 mm. Sn +0.25 mm. Cu +1 mm. Al	0.88 mm. Sn 0.25 mm. Cu +1 mm. Al	1.32 mm. Sn +0.25 mm. Cu +1 mm. Al	cm.	(13 mm. steel inherent filter) (1 mm. Pb+0.5 mm. Cu)	
Air	147	134	128	Air	108	
Surface	174	156	145	Surface	109	
2.5	192	173	160	2.5	113	
5.0	201	185	172	5.0	116	
10.0	216	196	188	10.0	118	
15.0	225	210	200	15.0	118	

are commonly used in therapy. The values with the uncoated bone chamber again expressed in percentage of those obtained with the lucite chamber are presented in Table VI and Chart III. Several interesting observations can be made. The radiation produced with 100 kv. (peak) and used either unfiltered or filtered with 1 mm. aluminum is gradually hardened until a depth of 10 cm. is reached. The measured values increase up to this depth as rays which do not penetrate the 0.5 mm. bone wall are filtered out by the tissues. A somewhat similar, though less pronounced, effect is observed for the 140 kv. (peak) radiation filtered with 2 mm. aluminum. When the rays are filtered with 0.25 or 0.5 mm. copper very little change in the hardness can be noted and the values remain at about 380 per cent; 220 kv. (peak) radiation filtered with 0.5, 1 or 2 mm. copper is softened to a certain extent but the highest values obtained at 15 cm. depth still remain below 283 per cent. The effect is more marked for the rays produced with 400 kv. (peak) and the values increase from 145 per cent at the surface to 200 per cent at 15 cm. depth when 1.32 mm. tin +0.25 mm. copper

filter is used. Even so the values remain below the 210 per cent at the surface for the 220 kv. (peak) radiation filtered with 2 mm. copper. Radiation produced with 1,000 kv. and filtered with 1 mm. lead and 0.5 mm. copper is not changed enough by the tissues to increase the bone ionization very much. From a value of 109 per cent at the surface it increases to 118 per cent at 15 cm. depth.

and 0.88 mm. tin.+0.25 mm. copper for 400 kv. (peak).

The main value of such measurements as here presented is their application to therapy. The damage to bone in the path of the beam during treatment ought to be proportional to the ionization produced in the bone. "Tissue dose" at any depth (or "depth dose") is ordinarily expressed in percentage of the skin dose. The ionization

TABLE VII

HALF-VALUE LAYERS OF THE RADIATION AT DIFFERENT DEPTHS IN A WATER PHANTOM; MM. COPPER

Depth	100 kv. (peak)	100 kv. (peak)	140 kv. (peak)	140 kv. (peak)	140 kv. (peak)
cm.	No filter	1 mm. Al	2 mm. Al	0.25 mm. Al	0.5 Cu
Air	0.05	0.09	0.17	0.42	0.58
0	0.05	0.09	0.15	0.38	0.4
2.5	0.08	0.10	0.18	0.35	0.5
5.0	0.10	0.12	0.24	0.35	0.5
7.5	0.13	0.14	0.26	0.35	0.5
10.0	0.15	0.15	0.35	0.35	0.5
15.0	0.15	0.15	0.35	0.35	0.5

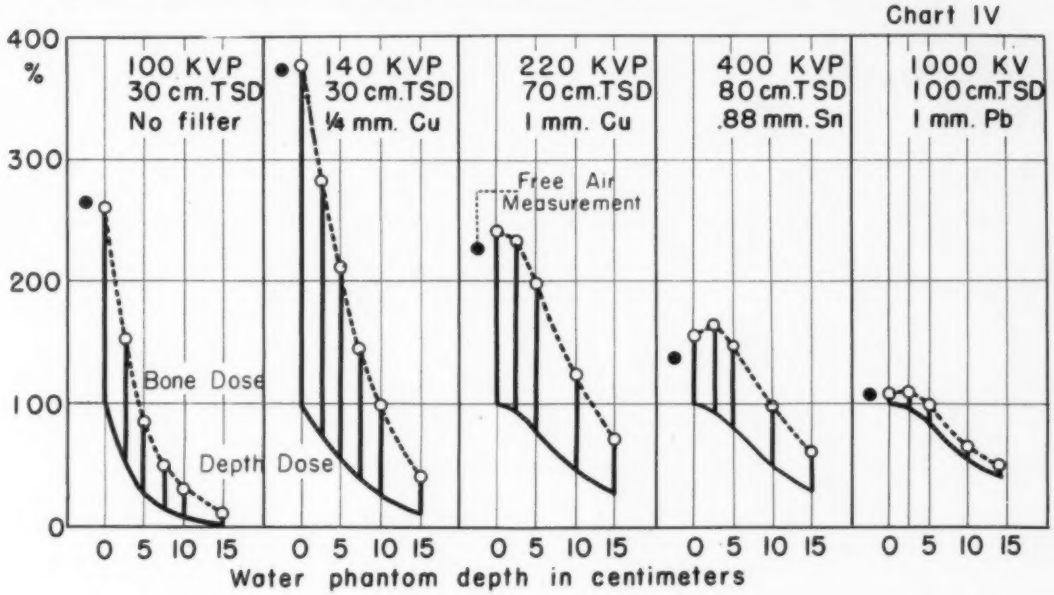
Depth	220 kv. (peak)	220 kv. (peak)	220 kv. (peak)	400 kv. (peak)	400 kv. (peak)	400 kv. (peak)
cm.	0.5 mm. Cu	1 mm. Cu	2 mm. Cu (or .44 mm. Sn)	0.44 mm. Sn	0.88 mm. Sn	1.32 mm. Sn
Air	1.34	1.90	2.44	3.8	4.4	4.8
0	1.2	1.6	2.1	3.0	3.45	3.9
2.5	1.1	1.5	2.0	2.3	3.0	3.4
5.0	1.0	1.4	1.9	2.15	2.6	3.1
10.0	0.95	1.2	1.6	1.9	2.2	2.5
15.0	0.9	1.1	1.5	1.7	2.0	2.2

These measurements give us some information about the extent to which the rays are softened at different depths in tissues. If it is assumed that rays with the same half-value layer contribute the same amount of secondary beta radiation from bone, then the half-value layer and equivalent wavelength for the radiation at different depths can be found from the obtained figures as shown in Table VII and Chart VI. This is not a very accurate method as pointed out by Clarkson and Mayneord[3] but it gives an approximation. It would seem from the figures obtained that a filter of 0.44 mm. tin+0.25 mm. copper (Thoraeus) may be advisable for 220 kv. (peak)

in bone may be expressed likewise in percentage of the skin dose. These figures on "bone dose" in percentage surface (or skin) dose, found in Table VIII and Chart IV, are obtained by multiplying the figures of Table VI by the corresponding percentage depth dose (Table VIII). For the same surface dose the bone dose is greatest when 140 kv. (peak) radiation filtered with 0.25 mm. copper (or a few mm. aluminum) is used, at least up to a depth of 5 cm. The 400 kv. (peak) filtered radiation supplies a smaller bone dose at depths less than 10 cm. As a general rule it can be stated that the harder the rays, the less damaging they are to bone if exception is made for the very soft rays

Depth Dose and Bone Dose in Percent of Surface Dose

Chart IV



Free Air Measurement

Dependence on Diameter of Chamber

Chart V

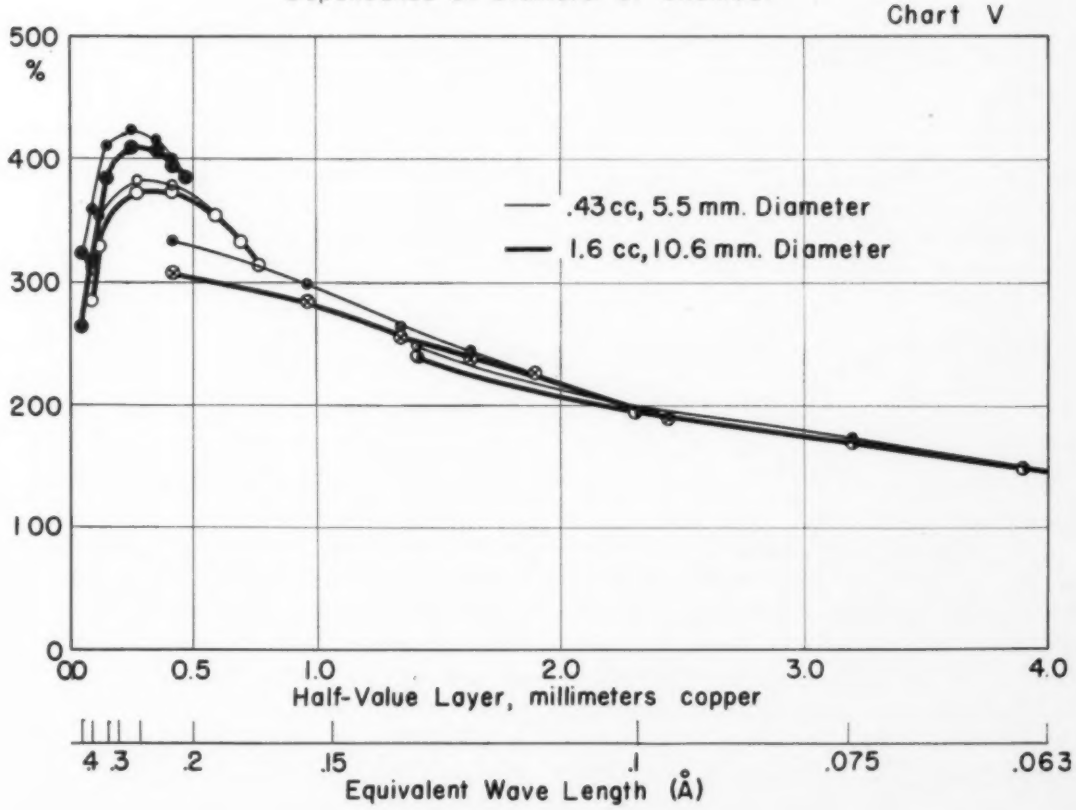


TABLE VIII

DEPTH DOSE (AIR WALL CHAMBER) AND BONE DOSE IN PERCENTAGE OF SURFACE DOSE

	100 kv. (peak)	30 cm. target distance
Depth	No Filter % Depth Dose	Half-value layer 1.7 mm. Al % Bone Dose
Air		263
Surface	100	260
2.5 cm.	51.5	153
5.0 cm.	25.4	84.5
7.5 cm.	14.1	50.2
10.0 cm.	8.1	30.9
15.0 cm.	2.7	10.5

	140 kv. (peak)	30 cm. target distance
Depth	0.25 mm. Cu Filter % Depth Dose	Half-value layer 0.42 mm. Cu % Bone Dose
Air		374
Surface	100	377
2.5 cm.	75.5	282
5.0 cm.	55.2	211
7.5 cm.	38.5	145
10.0 cm.	26.4	99
15.0 cm.	10.8	41.4

	220 kv. (peak)	70 cm. target skin distance
Depth	1 mm. Cu Filter % Depth Dose	Half-Value layer 1.9 mm. Cu % Bone Dose
Air		226
Surface	100	240
2.5 cm.	94.0	232
5.0 cm.	78.5	198
10.0 cm.	47.0	124
15.0 cm.	26.5	72

	400 kv. (peak)	80 cm. target skin distance
Depth	.88 mm. Sn Filter % Depth Dose	Half-Value layer 4.4 mm. Cu % Bone Dose
Air		134
Surface	100	156
2.5 cm.	95.0	164
5.0 cm.	80.0	148
10.0 cm.	50.5	99
15.0 cm.	30.0	63

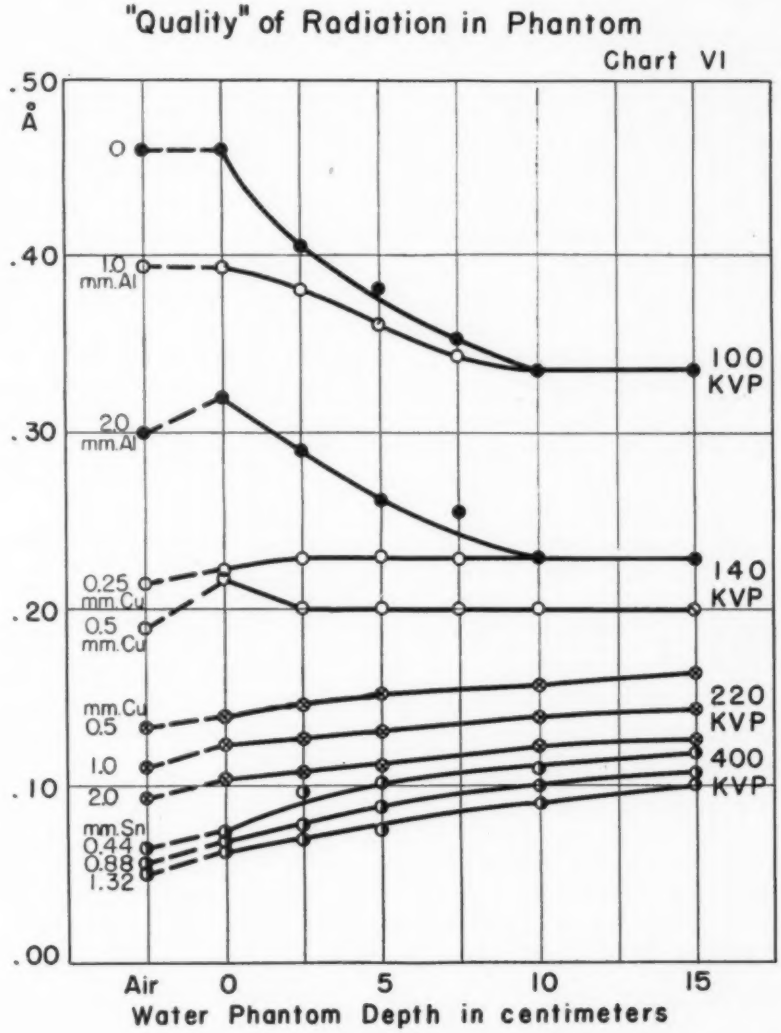
	1000 kv. 1 mm. Pb Filter	100 cm. target skin distance Half-value layer 9.5 mm. Cu
Depth	% Depth Dose	% Bone Dose
Air		108
Surface	100	109
2.5 cm.	97	110
5.0 cm.	86	100
10.0 cm.	55	65
14.0 cm.	43	51

which would have an intense effect only on the superficial layer of a bone at or very close to the surface.

The opposite rule applies, however, to the bone marrow as the secondary beta rays travel a very short distance, particularly for low voltages (few penetrate more than 0.01 mm.). The cortex reduces the intensity proportionately less the harder the rays.

Camp[2] reported recently on late bone necrosis occurring many years after roentgen therapy in a few patients who had been treated for pituitary tumors. These patients had been treated at a time (around 1920) when 140 kv. (peak) was the maximum voltage available. It is interesting to note

that the radiation used is the worst possible, as it supplies an extremely high bone dose near the surface where the temporal bone is located. Slaughter[10] has reported the most severe after effects (of 5 cases of fracture of the clavicle) in one case following the lowest "tissue dose" to the clavicle. It was noted that this case was treated with 140 kv. (peak) radiation, while the other cases reported were treated with 200 or 250 kv. or with radon. A number of reports are also found in the literature concerning fractures following irradiation of different bones (head of femur, ribs, clavicle).[4,6] The rays responsible for these results were as a rule somewhat harder but still in a range which supplies a high bone dose. Perhaps some damage of this type may be avoided with the use of rays of greater half-value layer.



SUMMARY AND CONCLUSIONS

Measurements have been made with ionization chambers constructed of bone in order to obtain some information on the total ionization in bone in connection with roentgen therapy. All ionization figures are expressed in percentage of bone chamber to "air wall" chamber of identical volume and capacity.

Maximum values of over 400 per cent (bone compared to lucite) were observed

for 100 kilovolts (peak) with a half-value layer of 0.3 mm. copper, and of about 380 per cent for 140 kv. (peak) with a half-value layer of 0.35 mm. copper. Values were lower for higher voltages and greater filtration.

Measurements taken with a water phantom indicated a greater amount of ionization in bone (up to 5 cm. depth in the phantom) when 140 kv. (peak) was used, than was observed at any other voltage. This may have direct application in therapy as 140 kv. (peak) may be preferred when ionization in bone is desirable, and used with caution when bone damage is to be avoided.

Figures are presented which show the change in the "quality" of the radiation as it penetrates the phantom. Radiation generated by the lower voltages is "hardened," and that generated by 220–400 kv. is "softened." One thousand kilovolt radiation is not altered appreciably in "quality" by the phantom.

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ABSORPTION MEASUREMENTS FOR BROAD BEAMS OF 1 AND 2 MEGAVOLT ROENTGEN RAYS*

By G. SINGER, C. B. BRAESTRUP†, *and* H. O. WYCKOFF

I. INTRODUCTION

ROENTGEN-RAY protection data are generally based on absorption coefficients determined for roentgen-ray beams of small cross section. Such coefficients can be accurately determined under laboratory conditions. Outside of the laboratory such narrowly collimated beams are not common. Therefore, it is to be expected that absorption data for collimated beams will not be directly applicable in designing protective barriers under conditions such that attenuation by the barrier is to an appreciable extent by scattering rather than by true absorption. These conditions obtain when the barrier material is of low atomic number and when the radiation is penetrating. For a heavy barrier material—such as lead—and for less penetrating radiation, attenuation is largely by true absorption. Only under these conditions may the absorption data obtained for collimated beams be safely applied to barrier design.

Concrete, being structurally self-supporting, relatively inexpensive, and easily fabricated, has become the standard material for the construction of barriers for protection against very penetrating radiation. Theoretical absorption curves applicable to such barriers are not currently available for wide-angle beams. In the calculations for such absorption curves a correction must be made for scattering which retains the scattered photons within the confines of the primary beam, a process tending to reduce the attenuation for wide-angle beams in comparison with that for narrow beams of the same radiation. This problem has been recognized in several papers relating to roentgen-ray protection.[1,2,3,4] However, because of the considerable technical difficulties involved in obtaining such data experimentally, all discussion in the literature relating to the increase in barrier thickness needed for wide-angle beams over that for narrow beams is of a general and indirect nature and not sufficiently detailed for specific application in the development of protection specifications.

In the design of protective barriers, information is also needed on the variation of roentgen-ray dosage rate with distance from the protective barrier to the position at which personnel will be stationed. Because of scattering from the wall it is to be expected that for very penetrating radiation this variation will not follow the inverse square law for the distance measured from the tube target. It is to be expected that this variation will be more rapid than indicated by this law because both the roentgen tube target and the irradiated volume of concrete act as sources of radiation.

It is the purpose of this paper to present (1) experimental data on absorption of roentgen rays in concrete for wide-angle beams generated by voltages of 1 and 2 megavolts, (2) preliminary data on the variation of the dosage rate with the distance from a protective barrier, and (3) data on concrete protection requirements for 1 and 2 mv. radiation under certain conditions which obtain in practice.

II. METHOD

The experimental arrangement used in obtaining the basic data given below is indicated in Figure 1. The concrete wall *C* is the main wall of the building housing the generator and is 41 inches* thick; *D* is the covered maze within which the ionization measurements were made.

* Because the thickness of protective barriers is often of practical interest to persons unfamiliar with metric units, we have specified the dimensions of all barriers in feet and inches.

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Dosage measurements were made by calibrated dosage instruments designed by Braestrup.[5] Three sizes of ionization chambers were used; their characteristics are listed in Table 1. The ionization chamber potentials were measured before and after irradiation by means of a string electrometer which is an integral part of the dosage meter. All ionization chambers were calibrated with both 175 kv. roentgen rays and gamma rays from radium. Both calibrations agreed very well with the theoretical values vased upon the air volume and capacitance. At the National Bureau of Standards the small thimble chamber and the electrometer head were checked against a similar instrument combination with 1 mv. radiation and were found to be in agreement therewith to within 2 per cent.

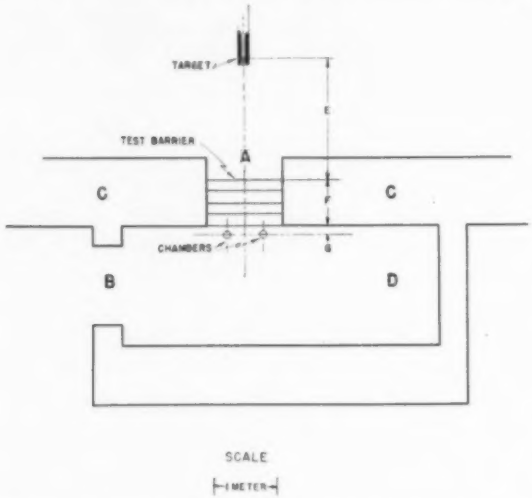

FIG. 1. Experimental arrangement showing two sets of concrete test blocks in place. A total of three such sets were used.

The barrier material consisted of solid concrete blocks of density 130 lb. per cu. ft. measuring $5\frac{3}{4}$ by $7\frac{3}{4}$ by $23\frac{3}{4}$ inches. These were laid without mortar in the doorway *A* shown in Figure 1. Measurements were made through sets of double layers of these blocks; one $5\frac{3}{4}$ in. thick and the other $7\frac{3}{4}$ in. The layers were staggered so that the planes of the cracks between blocks did not pass through either the tube target or ionization chamber. The opening remaining between the top of the test barrier and the lintel of the doorway to the maze was filled with lead bricks. For this arrangement both the radiation leakage through the doorway *B*—produced by the rays scattered through a glass-brick window above the main wall *C*—and that through cracks in the test barrier itself were found to be negligible in comparison with the dosage rate at the position of the test ionization chambers.

The distance *E* of Figure 1 was 1.83 meters in all cases; *F* is the barrier thickness. This thickness was converted to thickness of concrete of a density of 147 lb. per cu. ft. before plotting. The wall-to-center-of-chamber distance *G* varied from 2 to 4 inches for all plotted points. The ionization chambers were placed as near as practicable to the radiation barrier. It will be shown later that this position gives the minimum apparent absorption.

The roentgen-ray generator used is the 2 mv. roentgen-ray resonance generator described by Charlton and Westendorp[6]

TABLE I

IONIZATION CHAMBERS

Type	Wall Material	Inside Dimensions		Volume (cm.³)	r/Div.*
		Diameter	Length		
		cm.	cm.		
CX-1 Cylindrical	Bakelite	12.05	21.85	2490	0.23×10^{-3}
SX-11 Spherical	Polystyrene	10.5		624	0.79×10^{-3}
D-1 Thimble	Catalin	0.7	2.15	0.783	1.0

* Dose in roentgens per scale division of electrometer.

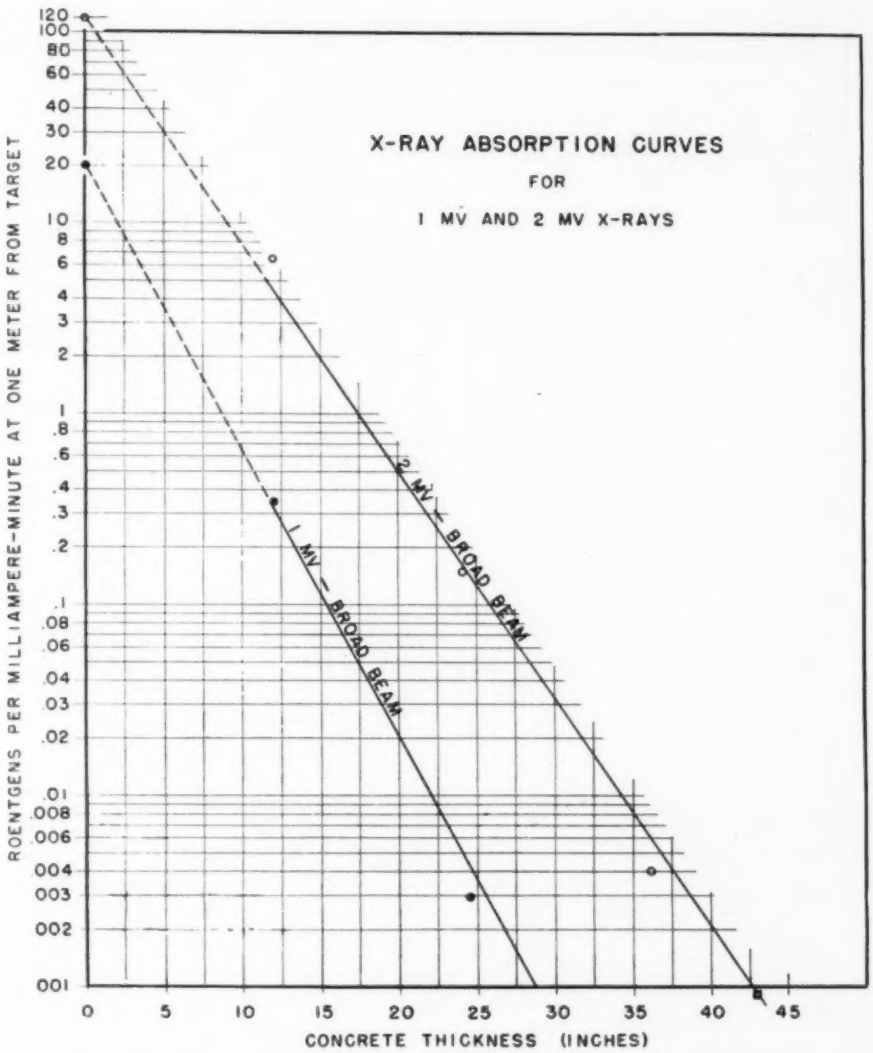

FIG. 2. Concrete absorption curves for 70° beams of 1 mv. and 2 mv. roentgen rays. All measurements were made with the radiation transmitted through a tungsten target along the tube axis. A resonance generator was used. The voltage waveform of such generators is sinusoidal. All indicated voltages are peak or crest values.

which is operative at both 1 and 2 mv.* All measurements were made with roentgen radiation transmitted through the tube target along the axis of the tube. The anode cone consisted of a cylinder with a lead wall 5 cm. thick, the end of which was shaped so as to confine the roentgen-ray beam to a cone of radiation measuring approximately 70° at its apex.

* We are indebted to the General Electric Company and to Dr. E. E. Charlton of their Research Laboratory for facilities which were made available to us in carrying out these experiments.

III. RESULTS

The attenuation curves obtained are shown in Figure 2. As a check on the reliability of the method used, transmission measurements for 2 mv. radiation through a solid concrete wall 42 inches thick of density 152 lb. per cu. ft. were also made. The single point so obtained is plotted in Figure 2 at a thickness of about 43 inches.

The effect of chamber-to-wall distance on the apparent attenuation is illustrated by the curves of Figure 3. The ordinate is

in arbitrary units. It is obtained by dividing the dose per milliampere-minute by the square of the sum of the distances E, F, and G. The abscissa is the chamber-wall distance, G, shown in Figure 1. If there were neither scattering in the wall nor absorption also for the 1 mv. data on a 4° beam published by Folsom and Focht.[2] Accordingly, it may be concluded that within these limits the beam angle has no important effect on the change in apparent attenuation with chamber-to-wall distance.

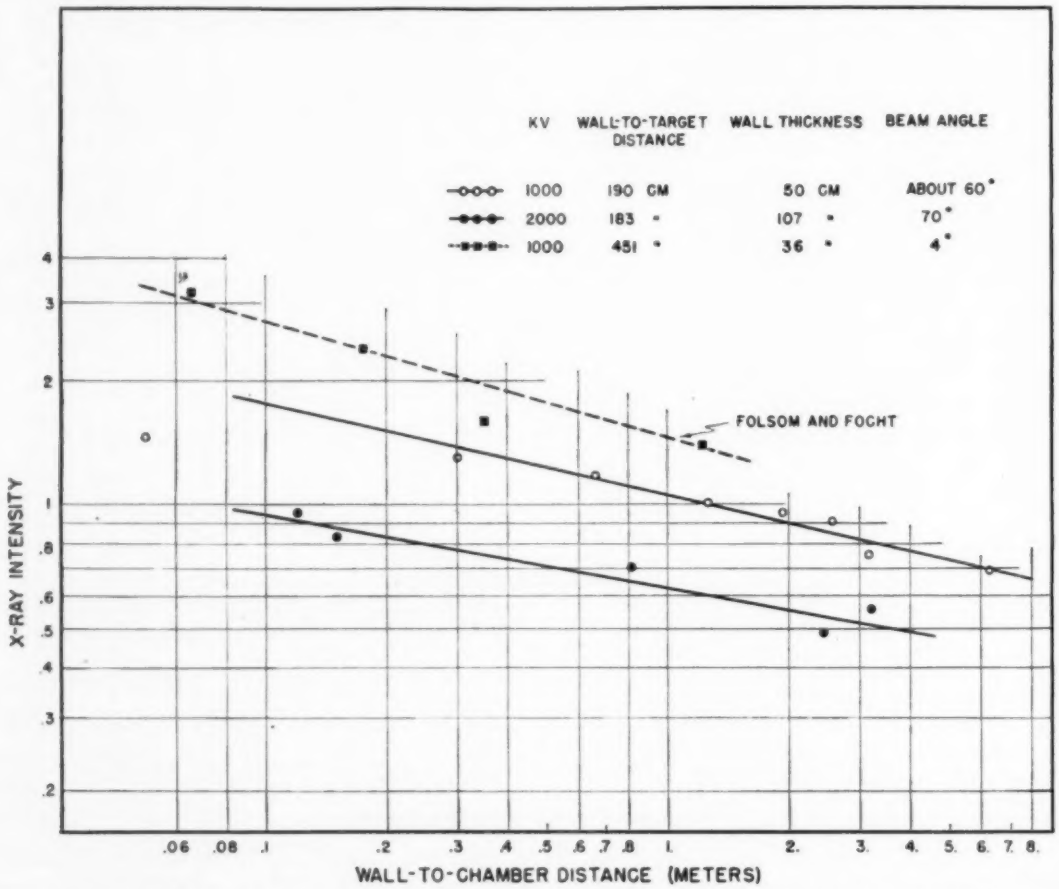

Fig. 3. Variation of roentgen-ray dose with distance from a concrete protective barrier. The abscissa is the distance to the ionization chamber measured from the side of the barrier from which the radiation emerges. The ordinate is obtained by dividing the dose per milliampere-minute by the square of the distance from the roentgen-ray source to ionization chamber.

in the air, each set of plotted points should give a straight line with zero slope. Actually, because of wall scattering, the value of the ordinate decreases with increase of abscissa. These plotted points fall along a straight line for each experimental setup. The slopes measured on a linear scale—not the logarithmic scale of Figure 3—are approximately 0.25. This is so not only for our own 1 mv. and 2 mv. data for 70° beams but

IV. Conclusions

From the data presented above it is possible to compute for broad roentgen-ray beams the thickness of concrete barriers necessary for protection against 1 mv. and 2 mv. roentgen radiation from targets of the transmission type. Such targets are standard in this quality range. In Table II the thickness of such barriers is listed for 1 mv. and 2 mv. roentgen radiation and for

TABLE II

THICKNESS OF CONCRETE OF DENSITY 147 POUNDS PER CUBIC FOOT REQUIRED FOR PROTECTION AGAINST BROAD BEAMS OF ROENTGEN RAYS FOR THE CURRENT, DISTANCE, AND VOLTAGE INDICATED

Target Distance	1 mv. Roentgen Rays				2 mv. Roentgen Rays			
Feet	0.5 ma.	1.0 ma.	2.0 ma.	3.0 ma.	0.5 ma.	1.0 ma.	2.0 ma.	3.0 ma.
	in.	in.	in.	in.	in.	in.	in.	in.
4	30.5	32.5	34.5	36.0	44.5	47.0		
5	29.5	31.5	33.5	34.5	43.0	45.5	48.0	49.5
6	28.0	30.0	32.5	33.5	42.0	44.5	47.0	48.5
8	26.5	28.5	30.5	32.0	39.5	42.0	44.5	46.0
10	25.0	27.0	29.5	30.5	38.0	40.5	43.0	44.5
15	23.0	25.0	27.0	28.0	35.0	37.5	40.0	41.5
21	21.0	23.0	25.0	26.5	33.0	35.5	38.0	39.5
30	18.5	20.5	22.5	24.0	30.0	32.5	35.0	36.5
40	17.0	19.0	21.0	22.0	28.0	30.5	33.0	34.5
50	15.5	17.5	19.5	21.0	26.0	28.5	31.0	32.5
75	13.5	15.5	17.5	18.5	23.0	25.5	28.0	29.5
100	11.5	13.5	15.5	17.0	21.0	23.5	26.0	27.5

the various tube currents and target distances normally encountered in practice. The tabular values given therein were computed directly from the absorption curves of Figure 2. A few points at short distances and high currents required linear extrapolation for their determination. Absorption of roentgen radiation by air has been neglected. A permissible daily dose of 0.1 roentgen has been assumed.[7,8] It was further assumed that the dosage rate per milliampere at 1 meter for a 1 mv. roentgen-ray generator is 20 r per minute and for a 2 mv. roentgen-ray generation, 120 r per minute, and that the person to be protected will normally be stationed near the protective barrier.

National Bureau of Standards
Washington, D. C.

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EDITORIAL

THE USE OF RADIOACTIVE ISOTOPES IN LEUKEMIA

DURING the past few years considerable attention has been focused on the study of leukemia, mainly because of the increasing availability of radioactive isotopes. In general, the biological effect of the radioactive isotopes is the result of the action of the emitted rays which are similar to those given off by radium. However, it was thought that due to inherent variation in the relative proportion of these rays and due to the many possible half-lives with the different isotopes, a new therapeutic agent could be found which would lead to irradiation of leukemia in a more efficient manner than is the case with radium or the roentgen rays.

The first attempts[1] were made with radiophosphorus (P^{32}). This element possesses properties which make it particularly attractive for investigations in leukemia. Aebershold[2] who produced radiophosphorus by deuteron bombardment of the stable red phosphorus in the cyclotron found the following important advantages: (1) radiophosphorus has a half-life of 14.2 days, the administered dose decaying in a few months to an imperceptible amount; (2) in contradistinction to radium which emits alpha, beta and gamma rays, it gives off only beta rays which have energies up to 1.8 mev., the average energy being approximately 0.6 mev., and (3) its chemical behavior is the same as that of ordinary phosphorus, thus taking an active part in the phosphorus metabolism of the body.

In 1942, Low-Beer, Lawrence and Stone[3] were already able to publish some definite data on the physical and biological background of the use of radiophosphorus and to give a brief account of its therapeutic possibilities in leukemia. Experimental studies in animals showed that radioactive phosphorus deposits with predilection in those tissues which normally have a higher phosphorus uptake, such as bone, spleen, liver and the lymph nodes. In leukemia, lymphoma and lymphosarcoma a greater percentage of radiophosphorus was concentrated in the pathologic tissues than in any of the normal tissues, except bone. As a rule, the greater the metabolic activity of a pathologic lymphoid cell, the greater was the uptake. It appeared that the more rapid synthesis of nucleoproteids in the actively growing cells required an increasing amount of phosphorus from the blood stream.

The fact that radiophosphorus emits only soft beta rays together with this rather selective deposition in pathologic lymphoid tissues suggested that an efficient procedure may be worked out for uniform internal irradiation of leukemia and allied diseases. In order to obtain an exposure over a longer period of time, Low-Beer, Lawrence and Stone[3] devised various methods. In using the simple saturation method, which is very much along the same line as Pfahler's saturation method for the roentgen rays, a large initial dose was given at the onset of the treatment and this was brought up to its original level every third day by making up the difference due to natural decay and loss by excretion. A second method was the fractional saturation in which a desired level was built up by small equal or by gradually increasing frac-

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[2] Aebershold, P. C. The cyclotron: a nuclear transformer. *Radiology*, 1942, *39*, 513–540.

[3] Low-Beer, B. V. A., Lawrence, J. H., and Stone, R. S. Therapeutic use of artificially produced radioactive substances; radiophosphorus, radiostrontium, radioiodine, with special reference to leukemia and allied diseases. *Radiology*, 1942, *39*, 573–597.

tions and maintained at the same height for a shorter or longer period. Finally, in a third method, which was called the fractional method, equal fractions of radiophosphorus were given at regular or gradually spaced intervals until the desired effect was produced. Considerable effort was spent in calculating the retained activity and the radiation level, or the maximum total exposure of the body, attained at the time of the administration of each dose. Although none of these methods led to a continuously uniform exposure and the radiation levels showed fluctuations due to the variation in the utilization of the doses given, clinically the fractional method with intravenous injection of the radiophosphorus proved a simple and convenient procedure. A review of the literature reveals that the great majority of the investigators in later work preferred the same procedure. The individual dose varied between 0.5 and 3 millicuries and was given once a week followed by gradually spaced intervals until the blood count became approximately normal.

The selective uptake of radiophosphorus by normal and leukemic tissues of humans was studied by a number of authors. Warren,[4] on the basis of postmortem examinations performed in 10 leukemic patients who died one to thirty-five days after the administration of the last dose of radiophosphorus, confirmed the increased concentration in all leukemia infiltrations. He also found that the liver, spleen, kidney and bone marrow contained relatively larger amounts of radiophosphorus than such slowly metabolizing tissues as brain, fat and cartilage, a fact already known from animal experiments.

In additional reports[5] Warren, from clinical observations, gave data and graphs of the retention and rate of excretion of the radiophosphorus. Essentially, they agreed with those of Low-Beer, Lawrence and Stone.[3] Finally, in 1945, he presented the results in 81 cases of leukemia, myeloma, Hodgkin's disease and polycythemia vera treated with radiophosphorus, most of which were far advanced or refractory to roentgen therapy. In 47 per cent of the patients with chronic myelogenous leukemia and 63 per cent of the patients with chronic lymphatic leukemia a temporary improvement was noted. The most important observation was that radiophosphorus produced radiation sickness only in rare instances.

A very comprehensive study on radioactive phosphorus as a therapeutic agent was published by Reinhard, Moore, Bierbaum and Moore.[6] These authors collected all the cases from the literature which were treated in one form or another with radiophosphorus and compared the results, as far as this was possible, with the results obtained in 155 cases of their own. In regard to myelogenous leukemia it was found that there were nine reports in the literature dealing with 107 cases and the authors themselves had 39. There was good agreement that in acute myelogenous leukemia radiophosphorus remained ineffective. In the chronic cases its administration restored the leukocyte count to normal or approximately normal in the majority of instances, a fact which was followed quite consistently by symptomatic improvement. The cases from the literature, with few exceptions, did not permit an evaluation of the final results but an analysis of the authors' series showed that in those patients who had already died the average duration from onset of symptoms to death was 3.64 years. In the subjects still living the average duration was 31.1 months, 3 cases having been treated for more than three years. The group of lymphatic leukemia included 120 cases collected from

[4] Warren, Shields. Distribution of doses of radioactive phosphorus in leukemic patients. *Cancer Research*, 1943, *3*, 334–336.

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ten reports in the literature and 45 personal cases. As in myelogenous leukemia, the acute cases showed no significant response. In the chronic cases, those who had already died had an average survival from onset of symptoms to death of 31.8 months and in the remaining the average duration of the disease amounted to 36.3 months. A comparison of these figures with data of older statistics of untreated leukemias or leukemias treated principally with radiation therapy is interesting. In 1924 Minot, Buckman and Isaacs[7] determined the average duration from onset of symptoms to death as being 3.04 years for chronic myelogenous leukemia and 3.33 years for chronic lymphatic leukemia, without treatment. In 1934, a collective review[8] of a rather large number of cases revealed that if roentgen (or radium) therapy was used the average survival from onset of symptoms varied between 3 and 4.1 years for chronic myelogenous leukemia and 2.75 and 4.33 years for chronic lymphatic leukemia.

The impression was gained by Reinhard, Moore, Bierbaum and Moore[6] that treatment with radiophosphorus in any type of chronic leukemia "is probably as satisfactory as, but no better than, X-radiation in relieving symptoms and in prolonging life." As in the observations of Warren, the chief advantage of radiophosphorus over roentgen therapy was found to be in the freedom from radiation sickness. However, roentgen therapy was noted to be more effective than radiophosphorus in bringing about a rapid regression of the enlarged spleen and lymph nodes. In such instances an association of the two therapeutic agents had definite merit. In the acute leukemias, as already stated, and in monocytic leukemia, radiophosphorus remained without value.

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During the course of these studies, it was soon suspected that the administration of radiophosphorus in leukemia must also have a harmful effect when there is already too much depletion of the bone marrow present or when the dose is too large. Thus Warren[5] observed that radiophosphorus was absolutely useless in all those instances in which but little hematopoietic tissue remained. Likewise Reinhard, Moore, Bierbaum and Moore[6] found that severe leukopenia, thrombocytopenia, and anemia may occur as complications of radiophosphorus therapy. As a rule, the leukocyte level decreased first, the thrombocyte level second and the erythrocyte level was affected last. There was a wide variation in the dose leading up to these complications, so that no accurately corresponding radiation levels could be established. The problem was further complicated by the difficulty of precisely assaying the activity of the radiophosphorus itself.

Graff, Scott and Lawrence,[9] to gain additional knowledge on the limiting factors of the use of radiophosphorus, performed a series of experiments with transmissible lymphoma in mice. As was expected the radiophosphorus localized with predilection in lymphomatous infiltrations, spleen and liver. It was found, however, that when the dose was raised to the point where it would destroy the lymphomatous infiltrations, all elements of the hematopoietic system, including leukocytes, thrombocytes and erythrocytes, were temporarily depressed. When the dose was raised further, the bone marrow was destroyed. From past experience with roentgen therapy it is known that cells following repeated irradiation acquire a certain radioresistance. It is probable, therefore, that in treating leukemia with radiophosphorus the stage is eventually reached when both leukemic and normal cell elements are being equally irradiated. This means that the threshold of selective action is not as high as was ori-

[9] Graff, W. S., Scott, K. G., and Lawrence, J. H. Histologic effects of radiophosphorus on normal and lymphomatous mice. Am. J. Roentgenol. & Rad. Therapy, 1946, *55*, 44–54.

ginally surmised. For the same reason, and since leukemia is such a diffuse disease, Graff, Scott and Lawrence[9] believe that the chances of finding a method of true selective irradiation are not very great.

Another type of isotope was tried by Thygesen, Vidabaek and Villaume.[10] These authors, in 1944, published a preliminary report on the treatment of leukemia with radiosodium (Na^{24}). The main difference between radiophosphorus and radiosodium is that the latter possesses no selective avidity but distributes itself through the extra- and intracellular fluid, maintaining an almost constant radiation level in the blood stream. Other distinguishing features are that the radiosodium emits beta rays and very hard gamma rays, and that its half-life is only 14.8 hours. Because of these factors, radiosodium acts more like "spray" roentgen therapy producing a rather uniform irradiation of the whole body. Thygesen, Videbaek and Villaume treated 7 cases of chronic lymphatic leukemia with apparently favorable results, but since the period of observation did not exceed 150 days no statement can be made as to the final value of the treatment. On the other hand, Lindgren,[11] who treated 5 cases of leukemia comparatively with radiosodium and radiophosphorus, found a rather poor response to radiosodium and a much better response to radiophosphorus. A very valuable report on the comparative study of the effects of radioactive sodium and of whole body roentgen irradiation on white mice was presented by Evans and Quimby.[12] A great similarity was noted between the two types of radiation suggesting that a like behavior occurs also in the human.

An excellent review was published recently by Furth[13] surveying all experimental aspects of the last few years, aiming to throw some light on the nature and genesis of leukemia. In summarizing the results of this review, Furth arrived at the conclusion that if diverse extrinsic and genetic factors act on a normal hematopoietic cell a transformation into a leukemic cell with a heightened growth force may follow. The basic change may be due (a) to a cytoplasmic or nuclear alteration of the reproductive material of the cell, (b) to the presence of a self-perpetuating exogenous agent, perhaps a virus, and (c) to an autocatalytically multiplying endogenous growth factor which causes abnormal differentiation. The leukemic cell, once on its way, exhibits complete independence of growth, invading tissues and organs and ultimately killing the host.

In the face of this latest theory and considering the limitations of all available forms of irradiation, it is probable that the curative treatment of leukemia will remain a problem difficult of solution for some time to come.

T. LEUCUTIA

[10] Thygesen, J. E. Videbaek, A. and Villaume, I. Treatment of leukemia with artificial radio-active sodium; preliminary report. *Acta radiol.*, 1944, *25*, 305–316.

[11] Lindgren, E. Versuche mit radioaktiven Isotopen bei Leukämiebehandlung. *Acta radiol.*, 1944, *25*, 614–624.

[12] Evans, T. C., and Quimby, E. H. Studies on the effect of radioactive sodium and of roentgen rays on normal and leukemic mice. AM. J. ROENTGENOL. & RAD. THERAPY, 1946, *55*, 55–56.

[13] Furth, J. Recent experimental studies in leukemia. *Physiol. Rev.*, 1946, *26*, 47–76.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: 1947, to be announced.

American College of Radiology
Secretary, Mac F. Cahal, 20 N. Wacker Drive, Chicago 6. Annual meeting: 1947, to be announced.

Section on Radiology, American Medical Association
Secretary, Dr. U. V. Portmann, Cleveland Clinic, Cleveland, Ohio. Annual meeting: 1947, to be announced.

American Radium Society
Secretary, Dr. H. F. Hare, 605 Commonwealth Ave., Boston, Mass. Annual meeting: 1947, to be announced.

Alabama Radiological Society
Secretary, Dr. John D. Peake, Mobile Infirmary, Mobile Ala. Next meeting time and place of Alabama State Medical Association.

Arkansas Radiological Society
Secretary, Dr. Fred Hames, 511 National Bldg., Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: Palmer House, Chicago, Ill., Dec. 1–6, 1946.

Radiological Section, Baltimore Medical Society
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

Section on Radiology, California Medical Association
Secretary, Dr. D. R. MacColl, 2007 Wilshire Blvd., Los Angeles 5, Calif.

Radiological Section, Connecticut Medical Society
Secretary, Dr. Robert M. Lowman, Grace-New Haven Community Hospital, New Haven 11, Conn. Meets bimonthly on second Thursday, at place selected by Secretary.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

Radiological Section, Los Angeles Co. Med. Assn.
Secretary, Dr. Roy W. Johnson, 1407 S. Hope St., Los Angeles, Calif. Meets on second Wednesday of each month at the County Society Building.

Radiological Section, Southern Medical Association
Secretary, Dr. Roy G. Giles, Temple, Texas.

Brooklyn Roentgen Ray Society
Secretary, Dr. A. H. Levy, 1354 Carroll St., Brooklyn 13, N. Y. Meets monthly on fourth Tuesday, October to April.

Buffalo Radiological Society
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

Chicago Roentgen Society
Secretary, Dr. T. J. Wachowski, 310 Ellis Ave., Wheaton, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

Cincinnati Radiological Society
Secretary, Dr. Samuel Brown, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. Carroll C. Dundon, 11311 Shaker Blvd., Cleveland, Ohio. Meetings at 6:30 P.M. on fourth Monday of each month from October to April.

Dallas-Fort Worth Roentgen Study Club
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meets in Dallas on odd months and in Fort Worth on even months, on third Monday, 7:30 P.M.

Denver Radiological Club
Secretary, Dr. W. C. Huyler, 1619 Milwaukee, Denver 6, Colo. Meets third Friday of each month at Department of Radiology, Colorado School of Medicine.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

Florida Radiological Society
Secretary, Dr. J. M. Dell, Jr., 333 W. Main St., S., Gainesville, Fla. Meetings in May and November.

Georgia Radiological Society
Secretary, Dr. James J. Clark, 478 Peachtree St., Atlanta, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Illinois Radiological Society
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. J. A. Campbell, Indiana University Hospitals, Indianapolis 7. Meets second Sunday in May.

Iowa X-Ray Club
Secretary, Dr. Arthur W. Erskine, 326 Higley Bldg., Cedar Rapids, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. Special meetings by announcement.

Kentucky Radiological Society
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

Long Island Radiological Society
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

Louisiana Radiological Society
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

Michigan Association of Roentgenologists
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

Milwaukee Roentgen Ray Society
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. Chauncey N. Borman, 802 Medical Arts Bldg., Minneapolis 2, Minn. Two meetings yearly, one at time of Minnesota State Medical Association, the other in the fall.

Nebraska Radiological Society
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Neb. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

* Secretaries of societies not here listed are requested to send the necessary information to the Editor.

New England Roentgen Ray Society
Secretary, Dr. George Levene, Massachusetts Memorial Hospitals, Boston, Mass. Meets monthly on third Friday, Boston Medical Library

New Hampshire Roentgen Ray Society
Secretary, Dr. A. C. Johnston, Elliott Community Hospital, Keene, N. H. Meets four to six times yearly.

Radiological Society of New Jersey
Secretary, Dr. W. H. Seward, Orange Memorial Hospital, Orange, N. J. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

New York Roentgen Society
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

North Carolina Radiological Society
Secretary, Dr. J. E. Hemphill, 323 Professional Bldg., Charlotte 2, N. C. Meets in May and October.

North Dakota Radiological Society
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

Central New York Roentgen Ra Society
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

Ohio Radiological Society
Secretary, Dr. Henry Snow, 1061 Reibold Bldg., Dayton, Ohio. Meets during annual meeting of Ohio State Medical Association.

Oklahoma State Radiological Society
Secretary, Dr. P. E. Russo, 230 Osler Bldg., Oklahoma City, Okla. Three regular meetings annually.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets first Tuesday of each month.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. J. M. Converse, 416 Pine St., Williamsport.

Philadelphia Roentgen Ray Society
Secretary, Dr. C. L. Stewart, Jefferson Hospital. Meets first Thursday of each month, October to May, at 8:00 P.M., in Thomson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. L. M. J. Freedman, 115 South Highland Ave. Meets 6:30 P.M. at Webster Hall Hotel on second Wednesday each month, October to May inclusive.

Portland Roentgen Club
Secretary, Dr. Selma Hyman, University of Oregon Medical School, Portland, Oregon. Meets monthly 2d Wednesday, 8:00 P.M., Library of University of Oregon Medical School.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Murray P. George, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary Dr. A. M. Popma, 220 N. First St., Boise, Idaho.

St. Louis Society of Radiologists
Secretary, Dr. Edwin C. Ernst, Beaumont Medical Building, St. Louis, Mo. Meets fourth Wednesday of each month, except June, July, August, and September.

San Diego Roentgen Society
Secretary, Dr. R. F. Niehaus, 1831 Fourth Ave., San Diego, Calif. Meets monthly, first Wednesday at dinner.

San Francisco Radiological Society
Secretary, Dr. Joseph Levitin, 516 Sutter St., San Francisco 2, Calif. Meets monthly on the third Thursday at 7:45 P.M., first six months of the year at Lane Hall, Stanford University Hospital, and second six months at Toland Hall, University of California Hospital.

Shreveport Radiological Club
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth 4, Texas.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets first and third Thursdays 4:00 to 5:00 P.M., September to May inclusive. Room 203, Service Memorial Institute, 426 N. Charter St., Madison.

Utah Radiological Conference
Secretary, Dr. Henry H. Lerner, School of Medicine, University of Utah, Salt Lake City 1. Meets 1st and 3rd Thursdays monthly from 7:30 to 10 P.M., Salt Lake County General Hospital, September to June.

Utah State Radiological Society
Secretary, Dr. M. Lowry Allen, Judge Bldg., Salt Lake City 1, Utah. Meets third Wednesday in September, November, January, March and May.

Virginia Radiological Society
Secretary, Dr. E. L. Flanagan, 116 E. Franklin St., Richmond, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Thomas Carlile, 1115 Terry St., Seattle. Meets fourth Monday each month, October through May, College Club, Seattle.

X-Ray Study Club of San Francisco
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly, third Thursday evening.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

Mexico

Sociedad Mexicana de Radiologia y Fisioterapia
General Secretary, Dr. D. P. Cossio, Marsella No. 11, Mexico, D. F. Meets first Monday of each month.

British Empire

British Institute of Radiology Incorporated with the Roentgen Society
Medical Members' meeting held monthly on third Friday at 2:30 P.M. and Ordinary Meeting at same time on following Saturday, October to May, 32 Welbeck St., London, W.1.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London.

Faculty of Radiologists
Secretary, Dr. M. H. Jupe, 23 Welbeck St., London, W.1 England.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital,

K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

Canadian Association of Radiologists
Honorary Secretary, Dr. E. M. Crawford, 2100 Marlowe Ave., Montreal 28, Que. Meetings January and June.

Société Canadienne-Francaise d'Électrologie et de Radiologie Médicales
Secretary, Dr. Origéne Dufresne, 4120 Ontario St., East, Montreal, P. Q.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

South America

Sociedad Argentina de Radiologia
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Sociedad Peruana de Radiologia
Secretary, Dr. Julio Bedoya Paredes, Apartado, 2306 Lima, Peru. Meetings held monthly except during January, February and March, at the Asociación Médica Peruana "Daniel A. Carrión," Villalta, 218, Lima.

Continental Europe

Ceskoslovenská společnost pro röntgenologii a radiologii v Praze
Secretary: MUDr. Roman Blána, Praha XII, Korunní 160, Czechoslovakia.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. Babaiantz, Geneva.
Secretary for German language, Dr. Max Hopf, Effingerstrasse 49, Bern. Meets annually in different cities.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banual Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad: USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

MINNESOTA RADIOLOGICAL SOCIETY

The fall meeting of the Minnesota Radiological Society was held in Rochester, Minnesota, on October 26, 1946. The following papers were presented:

Dr. S. F. Haines, Rochester. Radioactive Iodine in the Treatment of Exophthalmic Goiter.

Dr. J. J. Wells, Rochester. Unusual Case of Giant Cell Tumor of Bone Treated with X-ray.

Dr. E. T. Leddy, Rochester. X-ray Treatment of Malignancy of the Testis.

Dr. L. M. Vaughan, Rochester. Review of Different Methods of Radium Therapy for Carcinoma of the Cervix.

Dr. H. H. Bowing, Rochester. Essential Principles of Radium Therapy for Carcinoma of the Cervix.

Dr. R. E. Fricke, Rochester. Role of Alpha and Beta Particles in Radium Therapy.

Dr. M. M. D. Williams, Rochester. Loss of Radon from Radon Ointment.

Dr. John D. Camp, Rochester. Amebiasis in Naval Personnel.

Dr. Harry M. Weber, Rochester. A Case of Diffuse Lymphomatous Involvement of the Colon.

Dr. H. B. Burchell, Rochester. The Clinical Syndrome Associated with Pulmonary Arteriovenous Fistula.

Dr. H. B. Burchell and Dr. O. T. Clagett, Rochester. Motion Picture: A Case of Pulmonary Arteriovenous Fistula.

Dr. B. R. Kirklin, Rochester. The Diagnosis of Carcinoma of the Stomach by Chest Roentgenography.

Dr. C. Allen Good, Rochester. Thymic Tumor Associated with Myasthenia Gravis.

Dr. David G. Pugh, Rochester. Scleroderma of the Viscera.

Dr. John R. Hodgson, Rochester. Lesions of the Small Bones of the Feet: Neurotrophic or Infectious?

CANCER TEACHING DAY

A Cancer Teaching Day was held at Poughkeepsie, New York, on November 6, 1946. The following papers were given:

Dr. Chester O. Davison, Poughkeepsie. Five Year Cancer Survivals: The Experience of the Dutchess County Tumor Clinic.

Dr. John H. Farrow, New York. Diagnostic and Therapeutic Problems in Cancer: The Experience of the Dutchess County Tumor Clinic.

Dr. Martha E. Howe, New York. The Public Demand for Prevention and Early Diagnosis; A Sound Response.

Dr. George N. Papanicolaou, New York. Other Types of Cell Smear in Diagnosis of Cancer.

Dr. Maurice Fremont-Smith, Boston. The

Vaginal Smear in the Early Diagnosis of Cancer of the Uterus.

INDIAN JOURNAL OF RADIOLOGY

At the First Indian Congress of Radiology, held at Madras during February 13–16, 1946. it was decided to publish a journal entitled "*The Indian Journal of Radiology*" under the auspices of the Indian Radiological Association. This will be a quarterly publication. The editors, Dr. P. Ramma Rau and Dr. K. M. Rai, 155 Poonamallee High Road, Kilpauk, Madras, India, will be pleased to receive from radiologists original articles or case reports for publication in the Journal. The Journal will be published on art paper and it will therefore be possible to make a satisfactory reproduction of roentgenograms and other illustrations.

DAVID ANDERSON-BERRY PRIZE (1947)

A David Anderson-Berry Silver-Gilt Medal, together with a sum of money amounting to about £100, will be awarded during 1947 by the Royal Society of Edinburgh to the person who, in the opinion of the Council, has recently produced the best work on the therapeutic effect of roentgen rays on human diseases.

Applications for this prize are invited. They may be based on both published and unpublished work and should be accompanied by copies of the relevant papers.

Applications must be in the hands of the General Secretary, Royal Society of Edinburgh, 22 George St., Edinburgh 2, *not later than January 31, 1947*.

It should be noted that an extension of the period allowed for the receipt of papers has been made.

OKLAHOMA STATE RADIOLOGICAL SOCIETY

The Oklahoma State Radiological Society was organized on October 28, 1946. The following officers were elected: *President*, Dr. J. E. Heatley, Oklahoma City; *Vice-President*, Dr. W. E. Brown, Tulsa; *Secretary-Treasurer*, Dr. P. E. Russo, Oklahoma City. The society plans to hold three regular meetings during each year.

CLINICAL CONFERENCE OF MID-WESTERN RADIOLOGISTS

The Seventh Annual Clinical Conference of Mid-Western Radiologists will be held in Cleveland, Ohio, on Friday and Saturday, February 14 and 15, 1947, at the Stadler Hotel. An excellent program has been arranged with emphasis on the clinical aspects of radiology. Announcements of the meeting will be mailed to mid-western radiologists early in January.

DEPARTMENT OF TECHNIQUE

Department Editor: Robert B. Taft, M.D., B.S., M.A., 103 Rutledge Ave.
Charleston, S. C.

A SIMPLE PORTABLE WATER PHANTOM*

By JAMES F. MARVIN, M.S.
University of Minnesota
MINNEAPOLIS, MINNESOTA

THE water phantom which was constructed for use in "depth dose" and "bone dose" measurements has proved to be so easily and rapidly manipulated that a note on its construction seemed worth while. Less difficulty has been encountered in its use than with the customary presd-wood phantom. Special measurements with two or more ionization chambers of the condenser r-meter type and with bony structures as the pelvis or skull are easily made.

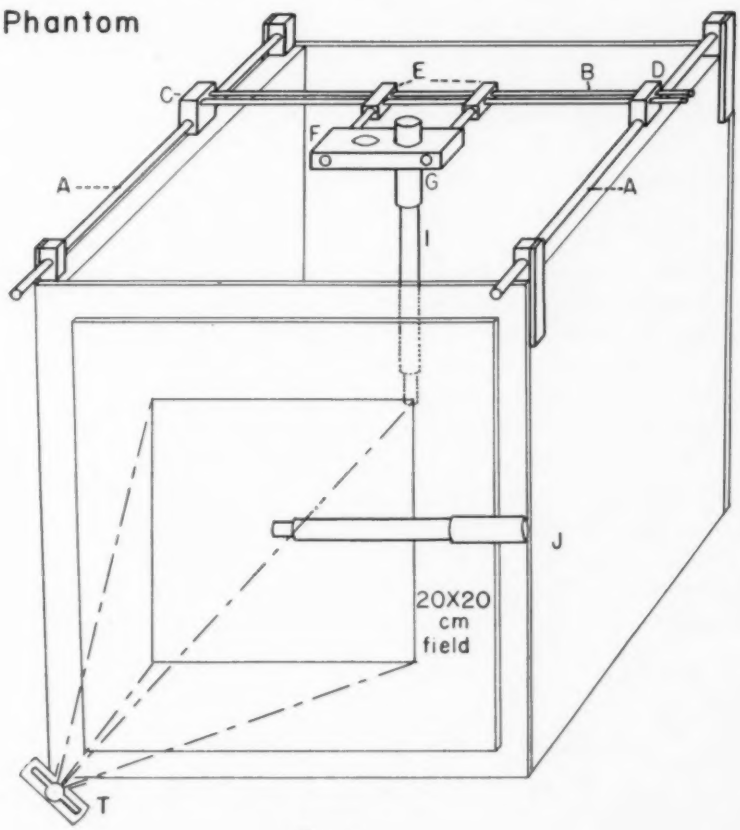

Fig. 1

Construction is clearly shown in the accompanying drawing. The box, 15×15×15

* From the Department of Radiology and Physical Therapy of the University of Minnesota and The University Hospitals, Minneapolis, Minn.

inches, has a wood base and sides. The two ends are of presdwood, which has a specific gravity of 1.03. A second sheet of presdwood is fastened on the front surface of the phantom. This sheet is cut to allow the ionization chamber to be placed half in presdwood and half in air for surface measurements.

The portions of the metal supporting structure for the chambers are: *A* and *B*, 5/16 inch steel rod; *C*, the hinge, which allows the rods *B* and other elements attached to *B* to rotate, removing the chamber *I* from the water; *D*, a stop, resting on rod *A*; *C* and *D* slide along *A* to allow for adjustment of depth in the roentgen-ray field. Clamps are put in place beside *C* and *D* on rod *A*, to maintain the same position for a series of readings. Supports *E* fasten on rods *B* at any desired lateral position in the field. Metal block *F* is designed to support two chambers in such a position that secondary radiation from one chamber will not seriously affect the reading of the second chamber. *G*, the cap of the r-meter chamber, may be moved vertically and clamped in place for any desired depth in the water. *I* shows the position of the ionization chamber in the water and *J* shows the position of the chamber for surface measurements. The two readings were not taken simultaneously as the absorption by the brass of chamber *J* may affect the reading of chamber *I*. Rather, a monitor chamber was introduced, with the thimble just penetrating the corner of the field. *T* represents the roentgen tube, shown in the drawing at 50 cm. distance from the surface of the phantom.

The chamber with the lucite thimble was found to be watertight. Other chambers may be made watertight with collodion, or may be immersed in a rubber sheath. It has been shown that this procedure does not alter the readings.

Leakage has occurred in the chambers in only two instances. Water affected the cement material used in the bone ionization chamber, and allowed water to enter the thimble. In the other instance, a chamber was accidentally dropped in the water. Amber and polystyrene are sufficiently nonhydroscopic that they are not affected by the water and did not develop leakage even when the chambers were left immersed in the water for forty-eight hours.

For depth dose measurements, a precaution should be noted. Measurement at the surface must be made with a dry chamber, and the temperatures of the water and air must be the same (or a correction may be made in the readings to take account of any temperature difference).

University Hospitals
Minneapolis 14, Minn.

REFERENCE

1. Stenstrom, K. W., and Marvin, J. F. Ionization measurements with bone chambers and their application to radiation therapy. Am. J. Roentgenol. & Rad. Therapy, October, 1946, *56*, 759–770.

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

HEAD

Wright, R. Wesley. Aero-otitis media; further report of purulent otitis media complicating aero-otitis media. *Ann. Otol., Rhin. & Laryng.*, Sept., 1945, *54*, 499–512.

The author reports on his observations concerning aero-otitis media as seen at an Army Air Base and Preflight School. Thirty-nine cases of purulent otitis media complicating aero-otitis media have been studied.

The ear symptoms experienced in flight are described as follows: During ascent there are usually no disagreeable symptoms. There may be a feeling of fullness and diminished hearing followed by a clicking sensation with clearing as air in the middle ear, now under greater pressure than the surrounding air, passes down the eustachian tube. When level flight is reached no further inequalities exist between the air in the middle ear and that of the outside, and no symptoms are experienced. It is seldom that this equalization is not accomplished on ascent. Descent is the critical time for the development of the ear symptoms. While any marked inequality between air in the middle ear and air of the surroundings will cause symptoms of stuffiness, pain, and hearing loss, actually this inequality occurs only on descent (except in rare cases).

Predisposing Pathological Changes Responsible for Aero-otitis Media. In general, upper respiratory infections, nasopharyngitis, sore throat, colds and sinusitis account for most of the acute conditions which are complained of as the predisposing cause. Of more specific interest are the findings in the epipharynx. The torus tubarius almost always shows marked inflammatory changes. The inflammatory swelling may obliterate the tubal orifice completely. Adenoid tissue, which is usually present in the vault, extends laterally and impinges on the torus tubarius. Usually there is little to be seen in the throat or anterior nose and the pathologic findings may be visualized only nasopharyngoscopically.

Fowler found a great number of flyers in England suffering from otitis media and aero-otitis media and in these patients, hyperplasia of the pharyngeal lymphoid tissue seemed to account for the condition.

Frequency. The incidence of aero-otitis media is about 6 per cent. By comparison, the incidence of aerosinusitis is a little over 1 per cent.

Symptomatology and Pathological Findings in the Ear.

1. Pain which may occasionally be so severe as to cause unconsciousness and shock.
2. Nausea, vomiting and dizziness.
3. A feeling of stuffiness usually supervenes after the pain disappears.
4. The drum is usually hemorrhagic and retracted if the pain is marked. When the process is moderate, there is only reddening along the handle of the malleus and around the periphery as in a beginning otitis media.
5. There is usually a moderate degree of hearing loss.
6. In practically every case of aero-otitis media there is sooner or later fluid in the middle ear. The fluid is a transudate drawn from the blood vessels of the middle ear as a result of the negative pressure.

Treatment.

1. Preventive—No difficulty should arise if no marked upper respiratory pathology exists and if the subject is aware of the possibility of ear blockage and is taught one of the various methods of opening the eustachian tube. Before the flight it is the practice of the author to have all men with any indication of upper respiratory infection ventilate the middle ear by the Valsalva method. The examiner watches the middle ear with an otoscope to see if the inflation is successful.
2. Treatment of the actual condition—The pain usually disappears within a short time. Inflation helps if stuffiness and hearing loss persists.
3. Treatment of the underlying pathology—The adenoid tissue seems to be the all important pathologic factor in the production of aero-otitis media. Those men having only respiratory disease without adenoid tissue rarely suffer from aero-otitis.

Therefore, to prevent future attacks of aero-otitis, the adenoid tissue is eliminated. If a large mass of adenoid tissue is found it is removed surgically. If the adenoid tissue is around the tubes and in the vault, which is its usual location, it is treated by irradiation in fractional doses. Radium has not been available to the author so roentgen therapy has been used exclusively. Those treated have responded well. Many men having repeated attacks of aero-otitis media have been freed of any difficulty on subsequent flights by this treatment.—*Mary Frances Vastine.*

Rosedale, Raymond S. Massive fibroma of the maxillary antrum as a part of multiple neurofibromatosis in siblings. *Arch. Otolaryng.*, Sept., 1945, *42*, 208–211.

Von Recklinghausen's disease or multiple neurofibromatosis is familial and sometimes congenital. In reviewing the inherited abnormalities of the skin in this disease, one investigator concluded that in a few families the defect is inherited as a dominant.

Clinical Picture. The disease is characterized by the association of multiple tumors of the nervous system (including the sympathetic, motor and sensory nerves). Sometimes there are associated abnormalities of the endocrine and vascular systems. Not infrequently café au lait spots of the skin accompany the disease.

Ewing reported associated defects, such as gliosis and tuberous sclerosis of the brain and spinal cord, spina bifida and meningocele. He also mentioned the association of various types of nevi and melanomas, localized and generalized elephantiasis and abnormalities of the bones. He specifically mentioned involvement of the mucous membranes. The superficial tumors are usually symptomless and become objectionable only because of their size, location, traumatization or multiplicity. More deeply seated tumors may cause symptoms because of anatomic tamponage occurring as a natural development in their growth.

Gross and Microscopic Pathology. Grossly the tumors are smooth, pearly gray and usually firm. They are easily separated from the surrounding tissues.

The tumors are usually multiple and their blood supply is poor. They grow slowly and are benign. They are subject to hyaline, fatty or mucoid degeneration. Calcification is sometimes seen.

Microscopically, the tumors consist, for the most part, of parallel and interlaced bundles of collagen and fibrous tissue with some tendency toward palisading of the nuclei.

Two case reports, one in a brother and the other in his sister, are presented. In both instances a massive maxillary fibroma was seen in association with Von Recklinghausen's disease. —*Mary Frances Vastine.*

Maliniac, Jacques W. Fracture-dislocations of the cartilaginous nose; anatomicopathologic considerations and treatment. *Arch. Otolaryng.*, Aug., 1945, *42*, 131–137.

The content of this article which would seem to be of interest to the radiologist is included under the following heading:

Mechanism of Fracture and Anatomic Changes. As a result of light trauma a fissure of the anterior part of the septal cartilage with formation of hematoma may take place.

Dislocation of the anterior part of the septum occurs under the impetus of trauma which strikes laterally at the mobile part of the nose. When the direction of the striking force is anteroposterior and the septal cartilage is caught in the sagittal plane, lesions are at maximum. Then the septal cartilage affixed in the vomer groove posteriorly may bend or fracture laterally in its anterior segment.

Two varieties of fracture of the septum are observed: vertical and horizontal. The vertical fracture of the septum is the more common. The line of fracture extends downward and backward from the dorsum toward the nasal spine, dividing the cartilage into two segments: the posterior, which is the larger, and the anterior, displaced in the frontal plane. The dorsal border of the anterior fragment pulls the nasal tip to the same side. Clinically, a vertical fracture reveals itself by the oblique position of the anterior segment of the septal cartilage inside one nostril. The anterior border of the septum, through pressure, causes displacement of the inner crus of the lower part of the alar cartilage, which thus protrudes under the vestibular skin.

When the upper lateral cartilage does not resist the impact of trauma, simultaneously with the vertical fracture a rupture occurs between nasal bone and cartilage, with or without marginal fracture. The latter often manifests itself in an exuberant bony callus.

The horizontal fracture of the septum usually runs obliquely downward and forward and a few

millimeters above the free border of the vomer. Diagnostically, it is often confused with subluxation of the septal cartilage from the vomer groove, which often occurs simultaneously with lateral displacement of the dorsum.

If infection does not take place, the fragments are unified by fibrous tissue or by a thin layer of cartilage, as in a green-stick type of fracture. Histologic examinations have shown that a post-traumatic bend of septal cartilage occurring in early life often results later on in ossification and distortion of the cartilage.

Complete subluxation of the septum on the vomer produces a double deflection of the dorsum: a depression caused by traction downward and backward and a lateral displacement in the direction of trauma due to detachment of the upper lateral cartilage, often with marginal fractures.

Fracture of the nasal bones frequently accompanies dislocation of the cartilaginous components. The nasal bones, which are thick in the upper half, thin down conspicuously toward their lower border and are intimately united along their inner borders, forming a solid bony block in the midline. Their thickness varies from approximately 7 mm. in the upper half to about 0.5 mm. at the lower border. This explains why in lateral trauma a fracture of the nasal bones never occurs in the midline but occurs usually along the points of least resistance, namely, at their junction with the frontal processes. In severe injuries, fracture occurs on both sides, and there is lateral displacement of the entire bony dorsum.—*Mary Frances Vastine*

NECK AND CHEST

Schilling, J. A. Struma lymphomatosa, struma fibrosa and thyroiditis. *Surg., Gynec. & Obst.*, Nov., 1945, *81*, 533–550.

Eleven cases are presented with clinical and pathological evidence to support their diagnosis. Six cases were diagnosed struma lymphomatosa; 2 cases were diagnosed struma fibrosa, and 3 cases represent the earlier, more acute, giant cell variant of struma fibrosa. Struma lymphomatosa is considered a distinct clinicopathologic entity. Its etiology is unknown, though it is considered a degenerative disease in contradistinction to a neoplastic or inflammatory disease. The excessive demands on the thyroid during the sexual life of the female are considered fundamental in the etiology of this disease. These excessive demands may be mediated through the hypophysis.

Struma fibrosa and its giant cell variant are considered two late manifestations of the thyroid from an acute to chronic inflammatory process. The fibrosis of the gland is considered first a replacement phenomenon of damaged glandular tissue. In later stages the diffuse infiltrating type of fibrosis may be due to a compensatory mechanism through constriction of the arteriolar blood supply of the gland, from a coincidental perithyroiditis.

Clinical Characteristics

1. *Age.* Acute thyroiditis occurs most frequently in young adults. The pseudogiant cell type of chronic thyroiditis and struma fibrosa occur in the third and fourth decades of life most frequently. Struma lymphomatosa occurs most often in the fourth or fifth decades.

2. *Sex.* Eighty-five per cent of the 3,750 patients admitted to the Strong Memorial Hospital, Rochester, N. Y., for thyroid disease were females. Thus the ratio of female to male with thyroid disease is roughly 6 to 1. The ratio of female to male in struma fibrosa is stated as roughly 3 to 2 or 4 to 1. In struma lymphomatosa almost 100 per cent of the patients are females.

3. *Predominant symptoms.* Symptoms are varied and often overlap, but, in general, the presenting symptom in struma lymphomatosa is *diffuse enlargement of the thyroid.* Occasionally mild pressure symptoms are noted such as dyspnea, dysphagia, hoarseness, voice change, tightness about the neck, cough or stridor. Signs and symptoms of early myxedema may be present.

With struma fibrosa, the symptoms are less vague than in struma lymphomatosa. *Pressure symptoms* are marked, often to such an extent that asphyxia seems imminent. Pain is usually absent.

With the giant cell variant of struma fibrosa, *pain* is usually the chief complaint and occurs in the neck or shoulder with radiation to the homolateral ear or back of the head.

4. *Response to roentgen treatment.* Struma lymphomatosa responds readily to roentgen therapy. This is in contrast to the lack of response of struma fibrosa. The response of pseudogiant cell chronic thyroiditis is unknown. Roentgen therapy may be used almost as a diagnostic test for struma lymphomatosa, provided that a preliminary biopsy has been taken to rule out malignancy.

5. *Postoperative course.* In general, with or

without operation, cases with struma lymphomatosa tend to become myxedematous and must be supported by thyroid extract. Struma fibrosa and its giant cell variant seldom cause myxedema provided that enough normal thyroid tissue has been allowed to remain after thyroidectomy. However, in struma fibrosa, pressure symptoms are apt to progress, and there is often a residual hardness toward the periphery of the lobes where resection was incomplete. The giant cell variant of struma fibrosa may subside spontaneously if left alone, though its symptoms may be so annoying that operation cannot be withheld.

6. *Laboratory.* Laboratory examination offers little diagnostic aid, with the exception of the basal metabolic rate which is usually normal or slightly elevated in struma fibrosa and its giant cell variant and low in struma lymphomatosa.

7. *Treatment.* Treatment of struma lymphomatosa by radiotherapy is the method of choice after a preliminary biopsy.

Surgery of struma fibrosa should be as conservative as possible. Its purpose is the relief of mechanical effects only as this disease is not neoplastic. Radiotherapy may be effective in the earlier, more acute, giant cell variant of struma fibrosa. The risk of operative complications of parathyroid tetany and recurrent laryngeal nerve paralysis is markedly increased in any radical attempt at extirpation of the gland and nullifies any potential relief of preoperative mechanical obstructive symptoms to the airway of the patient.

The end-result of treatment, barring surgical complications, is usually good, though with struma lymphomatosa there is a progressive tendency to myxedema.—*Mary Frances Vastine*

Carlton, Leffie M., Jr., and Adams, W. E. Resection of the lung in pulmonary suppurative diseases; factors contributing to its progress. *Surg., Gynec. & Obst.*, Dec., 1945, *81*, 623–630.

The results of lung resection in 59 cases of pulmonary suppurative diseases are presented. The factors which contributed to a satisfactory result were: rapid and complete expansion of the remaining lung, replacement of blood loss, avoidance of anoxia and prevention of serious infection following operation. The dissection technique of resection is the procedure of choice in most cases. Chemotherapy has been a major factor in the prevention of serious postoperative infection. There were 2 deaths in 45 operations made on 36 patients with bronchiectasis, 20 of which were bilateral.

In their discussion, the authors bring out the following points:

1. The surgical risk is considerably greater when there is bilateral involvement. Eight of the 20 bilateral group have had both operations completed, the course following the second operation being less difficult than after the first, This is partly due to the marked improvement that usually follows the first operation, thus making them better surgical risks, and also because little or no diseased lung remains after the second operation.

2. Clinical experience shows that human beings may tolerate the removal of a large percentage of the total lung capacity. Graham in 1940 reported the successful removal of both lower lobes, the right middle lobe and the lingula of the left upper lobe without the production of dyspnea or other physical handicap. In 2 of the authors' 8 completed bilateral operations, the same amount of lung resection was made. Both patients returned to full time work following surgery.

3. When bilateral operations are contemplated, sufficient time following the first operation for complete recovery and obtaining its beneficial effects should be allowed. This interval between operations may vary from six to twelve months or more depending on the occurrence of complications and the severity of the case.

4. The local and systemic use of sulfonamides in combination with the dissection technique is the procedure of choice in lobectomy for pulmonary suppuration. In 22 patients treated in this manner there were no bronchial fistulas and only 3 empyemas. Penicillin may replace the sulfa drugs as the chemotherapeutic agent of choice.

5. In pneumonectomy the pleural cavity is not drained following operation. Thus local sulfonamide therapy is less desirable because of the ill effects of rapid accumulation of fluid in the pleural cavity and collapse of the remaining lung. Penicillin, however, if deposited in the pleural cavity, may remain in effective concentration for twelve to twenty-four hours and without the production of an undue amount of pleural fluid.—*Mary Frances Vastine.*

McReynolds, George S., Jr., and Shelton, Fred W. Bronchography as a diagnostic aid in chest disease. *Ann. Otol., Rhin. & Laryng.*, March, 1945, *54*, 114–124.

The important factor in obtaining good bronchograms is the technique used. Iodochlorol was the opaque medium used by the authors since it is cheaper than, and apparently equally as efficient as, lipiodol.

Technique in Adults. Bronchography in adults is done under local anesthesia using 2 per cent pontocaine without epinephrine. The patients receive atropine and one of the barbiturates as premedication. The atropine is important since pontocaine has no inhibitory effect on the secretion of the mucosal glands and the barbiturates are used to offset any toxic effects of pontocaine.

A soft rubber catheter with a wire stylet is introduced into the glottis after which the stylet is withdrawn and the catheter passed down the trachea. The patient is then taken to the roentgen department where the catheter is manipulated into the proper bronchus under fluoroscopic visualization.

Technique in Children. After gas-ether induction, open drop ether is administered on the fluoroscopic table until the child is in deep third stage anesthesia. The anesthetic is then stopped. The larynx is exposed with a laryngoscope and the bronchoscope is introduced.

The authors usually fill both the right and left bronchial trees at one time to obviate the necessity of repeating the general anesthetic. However, one must be guided by the amount of pulmonary pathology present. When both lung fields are outlined, lateral films are of no value but oblique views are of great advantage.

With the patient in third stage anesthesia, the cough reflex is abolished and the breathing is slow and regular so that it is possible to get excellent films. One must have the complete cooperation of a competent anesthetist.

Contraindications. The authors have used the technique described in 20 children. No discernible ill effects have been noted. Several of the patients had such extensive suppuration that total pneumonectomy was required to effect a cure. It is felt that the aspiration of the bronchial tree before instillation of the iodized oil is an important factor in avoiding complications.—*Mary Frances Vastine.*

Holinger, Paul H., Hara, H. James, and Hirsch, Edwin F. Bronchogenic carcinoma. *Ann. Otol., Rhin., & Laryng.*, March, 1945, *54*, 5–36.

It is generally conceded that there has been not only an apparent but also an actual numerical increase in this disease. Bronchogenic carcinoma is fifth in frequency in types of carcinoma among males; it is surpassed only by carcinoma of the stomach, of the intestines and rectum, the prostate and bladder, and the liver and pancreas.

The present study is concerned with 175 proved cases of primary bronchogenic carcinoma observed principally at the Research and Educational Hospitals of the University of Illinois and at St. Luke's Hospital, Chicago, prior to February, 1944.

Etiology. It is generally agreed that no single agent is the sole cause but that a chronic irritant is an essential common factor.

Age, Sex and Race Distribution. Bronchogenic carcinoma is more frequently observed in white males. Seventy-two per cent of the authors' cases were between the ages of forty-five and sixy-four. The Negro male seems less susceptible to the disease than the white male.

Pathology. It is now believed that practically all carcinomas of the lung arise in bronchial tissue. Bronchogenic carcinoma shows a distinct predilection for the right bronchus.

The more common type occurs at or near the hilum.

Histopathologically, the commonest variety is the form composed of pavement epithelial cells and described as squamous cell.

Symptoms. The onset is always insidious. Almost all of the patients complained of disorders referable to the respiratory tract. Cough was the initial symptom in 50. Pain in the chest was the initial symptom in 14 patients.

Roentgenology. The principal feature roentgenologically is the extreme variation of the findings. An early tumor in a small peripheral bronchus frequently manifests itself only by an area of localized "pneumonitis" which appears entirely inflammatory in character. When such a lesion clears and then recurs, the probability that it is due to neoplasm is great.

Areas of pulmonary suppuration in older individuals, or otherwise unexplained pulmonary suppuration in a patient of any age, may well be due to pulmonary neoplasms.

A bronchus blocked by a tumor may become bronchiectatic in which case the bronchiectasis may be the outstanding roentgen finding.

The center of a solitary round tumor may become necrotic and evacuate itself to reveal a round shadow with the characteristic fluid level of a pulmonary abscess.

Thus it may be seen that the classical roentgen findings of bronchogenic carcinoma are those produced by varying degrees of bronchial obstruction. Small tumors on the wall of a bronchus produce no changes in most instances. Thus, negative roentgen findings in a patient who has occasional slight hemoptysis or who complains of a wheeze should not exclude further diagnostic studies, especially a bronchoscopic examination. Slightly larger tumors will manifest their presence on roentgenograms by signs of obstructive emphysema, the heart and mediastinum being shifted away from the affected side on expiration. Finally, total bronchial obstruction with collapse of the segment, the lobe, or the whole lung distal to the tumor is the most common roentgenologic manifestation of an intrabronchial neoplasm.

Bronchoscopy. The bronchoscopic picture is extremely variable. Usually the carcinoma is a soft, red, fungating papillomatous type of tumor which completely occludes a major bronchus.

Treatment. The treatment of bronchogenic carcinoma is primarily surgical. Roentgen therapy has received much consideration. In the authors' experience it has been of assistance in alleviating bronchial obstruction in some instances thus affording palliation.—*Mary Frances Vastine.*

Gross, Robert E., and Lewis, James E., Jr. Defect of the anterior mediastinum; successful surgical repair. *Surg., Gynec. & Obst.*, May, 1945, *80*, 549–554.

The substance of the anterior mediastinum is such that it offers relatively little resistance to displacement and hence it may bulge to either side. Herniation through the mediastinum is a well recognized phenomenon and has frequently been seen in conjunction with lesions which disturb the normal pressure relationships on the two sides of the chest. Under such circumstances a lung which bulges into the contralateral side of the thorax is always covered by its parietal pleura.

Distinct from subjects with a mediastinal hernia are those rare individuals in whom an actual communication exists between the two pleural spaces. This latter, congenital type of anatomical curiosity is probably best explained on the basis of an over-encroachment of the lungs in a forward and then medial direction during fetal life, so that the yielding anterior mediastinum becomes compressed and resorbed. If such encroachment is of only moderate severity, a septum-like type of anterior mediastinum could presumably result—such as that normally present in the dog. If, however, the encroachment is more advanced, the pleurae which appose one another may disappear and an opening of variable size can develop between the two cavities.

A large communication between the pleural cavities, per se, probably causes no physiological abnormalities or clinical symptoms. However, a mediastinal opening is a definite hazard if in addition there is some associated malformation or disease which disturbs the pressure equality which normally exists in the two sides of the chest. An example of such a mechanism is demonstrated by the authors' case.

Summary of Case Report. A four year old child had a large opening in the anterior mediastinum, a free interpleural communication and an anomaly of the right upper lobe bronchus. Deficiency of the bronchial cartilage allowed this airway to collapse in such a manner that air entered the upper lobe and became entrapped therein. Marked emphysema of the right upper lobe allowed it to protrude through the mediastinal opening and to invade the left pleural cavity. This child had intermittent attacks of severe respiratory distress, dyspnea, and cyanosis. These were completely relieved by simultaneous closure of the anterior mediastinal defect and removal of the anomalous right upper lobe. This is apparently the first recorded instance of surgical repair of an anterior mediastinal defect.—*Mary Frances Vastine.*

White, James C., Poppel, M. H., and Adams, Ralph. Congenital malformations of the first thoracic rib; cause of brachial neuralgia which simulates cervical rib syndrome. *Surg., Gynec. & Obst.*, Dec., 1945, *81*, 643–659.

The circulatory disturbances and neuralgias of the upper extremity which frequently result from cervical ribs or hypertrophy of the anterior scalene muscle are well understood. It is not generally appreciated, however, that a similar picture may be produced by a congenital abnormality of the first thoracic rib. The authors review the literature and add 10 new examples of this little known anomaly. Five of these have come to operation for relief of specific complaints, while 5 others have re-

mained wholly asymptomatic. Roentgenograms are reproduced of 8 of the 10 cases to illustrate different varieties of the deformity.

Summary. Congenital anomalies of the first thoracic ribs are not extremely rare.

First rib malformations generally consist of a rudimentary structure terminating in a synostosis or pseudarthrosis with the second rib near the scalene tubercle, or in a free end in the soft tissues at the base of the neck, which may be connected by a ligamentous band with the manubrium sterni. On very rare occasions the first rib may have a distinct joint near its lateral angle before it fuses with the second.

Other skeletal abnormalities are frequently present which cause further distortion of the thoracic outlet. They consist of deformities of the second rib, the upper end of the sternum, scoliosis of the cervicothoracic spine, and vertebral anomalies.

Congenital malformations of first thoracic ribs are best explained by errors of bodily segmentation in early embryonic development. These are brought about by abnormal formation of the brachial plexus and blood vessels, which make their appearance before the bony skeleton. Examples of posterior fixation of the brachial plexus and abnormalities in the arrangement of the arteries at the thoracic outlet were encountered in 2 of the authors' cases.

Symptoms and clinical evidence of abnormal first ribs consist of supraclavicular bony prominence, irritation or paralysis of the brachial plexus, and compression of the subclavian vessels as they cross the defective rib.

Cervical arthritis, early carcinoma of the thoracic apex, and herniation of the lower cervical intervertebral discs must be considered in the differential diagnosis of brachial neuralgia even in the presence of an anomalous rib.

A considerable proportion of first rib deformities are large enough to cause direct mechanical compression of the nerves and vessels at the thoracic outlet. When symptoms are attributable to this condition, conservative orthopedic measures should be tried before surgical intervention. Scalenotomy alone rarely suffices to decompress these structures, but must usually be accompanied by radical resection of the rib from a point close to its articulation with the transverse process forward to its attachment to the second rib or where it disappears beneath the clavicle. In cases in which an incomplete removal has been performed through the anterior supraclavicular approach, the central end of the rib may still cause irritation of the plexus. Resection of such a stump is then best accomplished through the posterior approach.—*Mary Frances Vastine.*

ABDOMEN

JONES, T. E., and KEHM, R. W. Total gastrectomy; report of eight cases. *Surg., Gynec. & Obst.*, May, 1945, *80*, 534–538.

Indications. Total gastrectomy is particularly adaptable for the linitis plastica type of stomach. However, it is also indicated in neoplasm extending into the cardiac end of the stomach and, even in deep penetrating ulcers located high in the stomach and therefore not amenable to medical management or to subtotal gastrectomy.

Physiologic Effects of Total Gastrectomy. The digestive disturbances most commonly encountered are diarrhea, steatorrhea, dysphagia and biliary regurgitation.

Alterations in the hemopoietic system after total gastrectomy are to be expected. Castle showed that pernicious anemia is at least partly due to loss of intrinsic factor from the stomach. Yet paradoxical as it may seem, the anemia is usually not pernicious but rather secondary or iron deficiency anemia of the normocytic, normochromic type.

Observations. Total gastrectomy is of extreme scientific interest and will open new fields for experimental physiologists. Questions which remain to be answered are: Why do these patients not develop pernicious anemia if the intrinsic factor is necessary to hemopoiesis? Why is the removal of the stomach followed invariably by steatorrhea and occasionally by diarrhea? What digestive disturbances follow loss of gastric enzymes?—*Mary Frances Vastine.*

POLAYES, S. H., and NEVINS, THOMAS F. Fatal hemorrhage from an angiomatous polyp of ileum complicating pregnancy. *Am. J. Obst. & Gynec.*, Aug., 1945, *50*, 207–212.

The literature contains reports of approximately 93 cases of angioma of the gastrointestinal tract. Kaijser collected and classified in table form 74 cases of angiomas of the gastrointestinal tract, of which 8 occurred in the ileum. In most instances, the angioma was one of several similar lesions co-existing in other segments of the intestines, especially in the jejunum. In very few was the lesion solitary and

limited to the ileum as in the case reported by the authors.

Discussion. Bleeding vascular lesions of the gastrointestinal tract may be due to telangiectasia (local or as an expression of a general diathesis), varicosities, or true neoplasms. The latter include cavernous angioma, capillary angioma, and angioendothelioma.

Grossly the lesions may manifest themselves in the form of ulcers, fungating bulky masses projecting into the lumen, or strictures.

In the case reported, a true neoplasm involving all layers of the portion of the ileum affected, was found. There was a marked tendency to nevus formation in the skin of this patient. There may have been a relationship to the angiomatous polyp in the ileum since many of the skin nevi were capillary angioma. Clinically it was noted that the pigmented nevi of skin became more accentuated and increased in number with pregnancy. It is conceivable that the state of gravidity activated the angioma as well as the skin nevi.

Kaijser mentions that in one of his cases of angioma of the stomach, bleeding increased with puberty. He mentioned puberty and gravidity among the endogenous stimuli which may activate the development of angiomas.—*Mary Frances Vastine.*

Griffin, William D., Bartron, George R., and Meyer, Karl A. Volvulus of the sigmoid colon; report of twenty-five cases. *Surg., Gynec. & Obst.*, Sept., 1945, *81*, 287–294.

Volvulus of the sigmoid colon is very uncommon in the United States. This is not true in Eastern Europe, for of 215 cases of intestinal obstruction reported by Pearlman from a Russian clinic, 111, or more than half, were cases of volvulus.

The authors reviewed the clinical and surgical records and the roentgen studies on 25 cases of volvulus of the sigmoid. The following conclusions were reached:

1. There are two main types of sigmoid volvulus: a. Acute (7 cases), characterized by occurrence in the younger age groups, short onset, equivocal history of constipation, early transient emesis, generalized cramping abdominal pains, abdominal tenderness, acute distention, and marked prostation. These patients tend to develop gangrene early and run a fulminating course. b. Subacute (18 cases), characterized by occurrence in the older age groups, of a more gradual onset of symptoms, history of previous attacks and constipation, and emesis late in the course of the disease. These patients tend to develop gangrene slowly and run a more moderate course.

2. Seventy-six per cent of the patients were unable to take an enema of more than 500 cc.

3. The single most helpful aid was roentgen examination. The typical roentgenographic findings are: tremendously dilated sigmoid loop situated in the right abdomen; moderate distention of the colon above the volvulus; absence of a collection of fluid within the bowel; "ace of spades" appearance of the barium enema opacity. (The barium enema shows a normal mucosal pattern in the sigmoid and rectum distal to the dilated loop. The upper end of the opacity, however, comes to a sharp point and produces the appearance of an "ace of spades".)

4. Volvulus of the sigmoid tends to recur and therefore simple detorsion is not the treatment of choice.

5. Exteriorization and second stage resection give the best results.—*Mary Frances Vastine.*

Hawe, Philip. The surgical aspect of intestinal amebiasis. *Surg., Gynec. & Obst.*, Oct., 1945, *81*, 387–404.

The object of this paper is to review the types of intestinal amebiasis which frequently arise in surgical practice in endemic areas; distant complications, such as hepatic abscess, are not included. It is based on experience gained during four years of hospital work in the tropics and on a study of the literature. The observations are supplemented with illustrative examples from a series of 450 cases of intestinal amebiasis admitted to a British General Hospital in South India between July, 1942, and March, 1944.

Summary and Conclusions

1. Intestinal amebiasis is the disease of major surgical importance in endemic areas. With the return of the forces from the East, examples of this disease must be expected in general practice.

2. The clinical and roentgenological manifestations of the localized forms of intestinal amebiasis may be indistinguishable from those of surgical diseases, acute or chronic. It is usually, but not always, possible to find the *Endamoeba histolytica* in the stools; sigmoidoscopy may succeed when examination of the

stools fails. The response to emetine is of considerable diagnostic significance, but occasionally the condition is resistant to the drug, and exploration or biopsy is then required. The possible coexistence of amebiasis with other lesions must not be overlooked.

3. Perforation of an amebic ulcer is to be expected in less than 3 per cent of cases. It is most often associated with fulminating infections. Toxemia may mask the clinical signs, and if gangrene is present recovery, even with operation, is very unlikely.

4. The clinical features of acute cecal amebiasis and acute appendicitis have much in common; the differential diagnosis may present great difficulty, but in view of the special risks of operation in cases of cecal amebiasis, every endeavor should be made to obtain a clinical diagnosis. If exploration proves necessary, manipulation of the cecum should be avoided as far as possible, and, unless the appendix is obviously in a dangerous condition, it should not be removed if amebiasis is found; emetine should be given at once after operation.

5. However suggestive of acute appendicitis, operation should not be advised in any case with an inflammatory mass until cecal amebiasis has been excluded. Amebic appendicitis is regarded for purpose of diagnosis and treatment as an extension of cecal amebiasis—unless complicated by abscess or perforation, it is a medical disease.

6. Unsuspected intestinal amebiasis and post-dysenteric conditions of non-appendicular origin often give rise to symptoms very suggestive of chronic appendicitis. Before appendicectomy is advised, these conditions must be carefully excluded. Operation is occasionally required for residual appendicular disease following cecal amebiasis.

7. Examples of localized chronic amebic colitis and ulceration of the rectum which may be mistaken for carcinoma or other surgical diseases are common in endemic areas. Minor anorectal conditions, such as piles, fissure and fistula, may result from unsuspected mild or chronic amebiasis.

8. With few exceptions, abdominal or rectal operations are strongly contraindicated in patients suffering from intestinal amebiasis—they are often followed by serious complications peculiar to this disease. If operation is necessary, or if amebiasis is discovered at exploratory operation, the sooner the emetine is commenced the better. Appendicostomy and cecostomy have no place in the treatment of this condition.—*Mary Frances Vastine.*

GYNECOLOGY AND OBSTETRICS

Willson, J. Robert. Carcinoma of the cervix complicated by pregnancy. *Am. J. Obst. & Gynec.*, Sept., 1945, *50*, 275–283.

Six patients with carcinoma of the cervix and pregnancy have been treated at the Chicago Lying-in Hospital since 1931.

Diagnosis. Bleeding which appears during early pregnancy and which persists for more than one week despite bed rest may be due to a malignancy. There are no subjective symptoms in the early case of carcinoma of the cervix. The signs during pregnancy are exactly like those which characterize the same lesion in the non-pregnant uterus: abnormal discharge or bleeding which, characteristically, is painless, irregular, progressive in amount, and prone to follow trauma.

Prognosis. The prognosis for carcinoma of the cervix discovered during pregnancy and properly treated seems to be at least as good as that in the non-pregnant individual. The belief that the rate of growth of carcinoma of the cervix is increased by the pregnancy has not been substantiated. Peller, after demonstrating that the relative number of carcinomas occurring during pregnancy was significantly less than the number occurring in similar age groups in the non-pregnant, concluded that pregnancy not only increases the resistance to cancer, but raises the average age of its occurrence and retards its progress.

It has been suggested that the marked rise in the estrogenic hormones during pregnancy may be a factor both in the production and extension of the disease. However, no malignancies developed in a group of animals studied by Emge and he concluded that the rôle of the estrogenic hormones in the production of cancer is secondary to, and limited by, the hereditary factors.

The Fetus. If external irradiation is given over the uterus during early pregnancy and abortion does not follow, fetal abnormalities occur in a high percentage of cases. Microcephalic idiocy has been the defect most commonly noted. Murphy advises artificial interruption of any pregnancy which continues after irradiation.

Treatment.

1. First trimester—Roentgen therapy should

be started as soon as the diagnosis is established. If abortion does not occur (usually during the third week), curettage is done. External irradiation (3,000 r to each parametrium) is followed by radium insertion after the uterus is empty. The total radium dosage consists of at least 5,000 mg-hr. of which one-third is delivered to the uterus and two-thirds to the cervix and surrounding parametrium.

2. Second trimester—Early in this trimester, treatment is as above but the interruption of pregnancy is less consistent. Effective irradiation to the pelvis is therefore diminished because of the increased size of the uterus.

During the late second trimester, an initial surface interstitial and/or intracervical application of the radium may be followed, after the reaction has subsided, by cesarean hysterectomy. A complete course of roentgen therapy, which may be started as soon as the wound is healed, is followed by the intrauterine application of radium to complete the total dosage. Delivery from below is contraindicated unless the lesion is very small.

3. Third trimester—The treatment in this period is much like that during the latter part of the second trimester. There is no reason for delaying the initial treatment of the cancer. In the majority of cases, abdominal delivery is the method of choice.

Summary. A high fetal mortality is inevitable since it is usually unwise to resort to methods of treatment which attempt to preserve the fetus but which may decrease the chances for cure of the neoplasm.—*Mary Frances Vastine.*

Melody, George F., Faulkner, Robert L., and Stone, Sidney J. Adenoacanthoma of the ovary. *Am. J. Obst. & Gynec.*, May, 1945, *49*, 691–695.

The term adenoacanthoma is applied to tumors composed of glandular and squamous elements. Descriptions of such lesions encountered in the esophagus, stomach, gallbladder, pancreas, sigmoid, thyroid and lung have been reported sporadically in the literature. The fact that occasionally, carcinoma of the corpus uteri are composed of both glandular and squamous cells is well known. The authors have been unable to find any case reports of primary adenoacanthoma of the ovary. Two such cases are presented by them.

Discussion. It is the conclusion of Novak that the "epidermization" as seen in fundal adenoacanthoma is the result of metaplasia of the cylindrical to the squamous type of epithelium. A study of cervical biopsy material reveals the very great frequency of metaplasia of the normal columnar to the squamous type of epithelium in inflammatory lesions of the cervix uteri. Moreover, it is recognized that the germinal epithelium covering ovaries which are involved in chronic perioophoritis occasionally undergoes squamous metaplasia. Then, there is the condition known as cystic fibrosis of the pancreas in which, due to the impaired absorption of fat-soluble vitamin A, there is a pronounced squamous metaplasia of the bronchial mucosa. It would appear, therefore, that squamous metaplasia is really of rather frequent occurrence in a wide variety of pathologic processes, running the gamut from chronic inflammatory lesions, and a vitamin deficiency state, to frank neoplasms. It seems entirely tenable, therefore, to assume that the squamous elements in the ovarian adenoacanthomas herein presented are, in fact, the result of the ubiquitous process of metaplasia.

Another plausible hypothesis for the presence of squamous epithelium in ovarian carcinoma is the supposition that such may result from Walthard's islands. According to Ewing, the latter are thought to result from "invaginations of celomic epithelium." The manifold possibilities of celomic epithelium on differentiation are well known. It may well be that the origin of the squamous elements in ovarian acanthoma is identical with the generally accepted theory of histogenesis of Brenner tumor, viz., Walthard's rests.—*Mary Frances Vastine.*

Hodgson, Jane E., Dockerty, M. B., and Mussey, R. D. Granulosa cell tumor of the ovary; clinical and pathologic review of sixty-two cases. *Surg., Gynec. & Obst.*, Dec., 1945, *81*, 631–642.

In their summary and conclusions, the authors include the following observations as a result of their study of 62 granulosa cell tumors.

1. These 62 granulosa cell tumors removed surgically between 1910 and 1944 constitute 1.63 per cent of the ovarian tumors (3,800) encountered during this period. About 60 per cent of these tumors were found in women who had passed the menopause.

2. The most common clinical symptoms were uterine bleeding (74 per cent), amenorrhea (22 per cent), and abdominal enlargement (29 per cent). The tumors that produced these symptoms grew slowly and might have been present

for as long as thirty-five years.

3. Urinary assays in one case (a woman aged fifty-seven) were positive for 8 rat units of estrogen per liter of urine excreted during the first twenty-four postoperative hours. The excretion dropped to zero during the next twenty-four hours.

4. Further evidence of hyperestrinism in this series is afforded by symptoms of precocious puberty, amenorrhea, and postmenopausal bleeding and is supported by the incidence of adenomyosis and endometriosis (0.6 per cent) which often occurred in post-menopausal patients; uterine fibromyomas (51.6 per cent); uterine hypertrophy (59.6 per cent) and proliferative endometrium (67 per cent).

5. There is a suggestion that granulosa cell tumors may produce the differentiative hormone, progesterone.

6. Twenty-one per cent of the 38 postmenopausal patients who had granulosa cell tumor also had endometrial carcinoma. In 3 of these cases, carcinoma of the breast also developed. This phenomenon of coexistent ovarian, endometrial, and mammary carcinoma in the human being bears a marked similarity to the results of experiments on laboratory animals in which estrogen stimulation appears to be a factor in carcinogenesis.

7. Pathologically, the tumors appeared to be of a low order of malignancy as determined by histologic grading.

8. Chemical analysis of four granulosa cell tumors indicated an excess of cholesterol and cholesterol esters as compared to the normal content of these substances in the ovaries. In general, this increase in lipoid content was thought to be related to storage of estrogenic hormone.

9. In this group of 62 cases of granulosa cell tumor there were 4 definite recurrences and 1 possible recurrence. The appreciable incidence of recurrences among older patients indicated that bilateral oophorectomy with hysterectomy is advisable for granulosa cell tumors affecting women past middle life. Less radical procedures, on the other hand, seem to be indicated for granulosa cell tumor among younger women inasmuch as no recurrences were noted and pregnancy subsequently occurred in 3 cases.

10. The authors' experience with postoperative irradiation for granulosa cell tumors in postmenopausal patients is not sufficient to warrant recommendations concerning this mode of treatment. However, it cannot be stated that such treatment might not be of value. Preoperative irradiation of the tumor may produce temporary regression with cessation of postmenopausal bleeding. If granulosa cell tumors are to be cured by irradiation, apparently the dosage must be markedly increased, if feasible, above the amount hitherto employed.—*Mary Frances Vastine.*

GENITOURINARY SYSTEM

MOE, RUSSELL J. Duplication of right kidney pelvis and ureter with extravesical ureteral opening. *Am. J. Obst. & Gynec.*, May, 1945, *49*, 641–646.

Ectopic ureters are found more frequently in females than in males, the ratio being about 2:1. The reason for this is that in the female an ectopic ureter usually opens distal to the vesical sphincter and produces a dribbling incontinence in females is found present since birth, but in spite of this there is normal emptying of the bladder.

Diagnosis. A dribbling incontinence of urine in the presence of normal bladder function is pathognomonic of ectopic ureter when occurring in a female. In the majority of instances this symptom has been present as long as the patient can remember. In a few reported instances the incontinence is present only when the patient is in the upright position.

Much patience and diligence are often necessary in locating the orifice of the ectopic ureter. An intravenous pyelogram which shows multiple pelvis on one side, and this in the presence of incontinence, should make one suspicious of an ectopic ureter.

According to Weigert's law, the ureter draining the upper part of the kidney opens lower in the urinary tract and it is always this orifice which is ectopic.

Treatment. The functional value of the kidney on the opposite side should be determined before any treatment is attempted.

Heminephrectomy seems to be the procedure of choice where the segment of kidney drained by the ectopic ureter is small and therefore functionally of little value.

The accessory kidney pelvis and ureter are usually infected so implantation of the accessory ureter into the bladder does not seem to be a wise procedure.—*Mary Frances Vastine.*

PRATT, J. P., and SCHAEFFER, ROBERT L. Sex precocity, virilism, adrenal cortical tumor.

Am. J. Obs. & Gynec., May, 1945, *49*, 623–633.

Classification of sex precocity is difficult because no two cases reported are exactly alike. The alteration in development may be due to (1) endocrine hyperfunctional tumors (adrenal, ovary, testis), (2) endocrine hyperfunction with or without cellular hyperplasia, (3) tumors in the region of the third ventricle, and (4) pineal tumor.

Sex precocity is not rare. In survey of 2,311 delinquents in the preadolescent and adolescent age groups, it was revealed that sex precocity occurred in thirteen, or 6.8 per cent of them. Three case reports are given:

Case 1. A girl, aged two years nine months, who showed marked signs of virilism, was operated upon and an encapsulated tumor in the right adrenal was removed. At the end of a period of six years there have been no signs of metastasis. There has been marked reversal from the original picture to that of the normal female child.

Case 2. A female child at four months of age presented signs of virilism which increased until near her death at the age of twenty-five months. The first lesion noted was fibrosarcoma of the cheek. This metastasized to the left adrenal which accounted for the signs of virilism. The death of the patient was due to the malignant tumor of the cheek and its metastases. The virilism appeared as a consequence of metastases to the adrenal.

Case 3. This girl, aged eight years four months, displayed signs suggesting virilism. Adrenal tumor was suspected, but exploratory operation revealed no adrenal enlargement. The discrepancies, when compared with the other two patients, were lack of increased statural growth, no change in voice, and only moderate enlargement of the clitoris. Her behavior was masculine.

Discussion. Sex precocity is more frequent in the female. Virilism in the female signifies sex reversal and is comparable with sex precocity in the male.

In his discussion of this paper, Dr. Novak brings out the following points: (1) The distinction which gynecologists most frequently must make in the cases of precocious puberty is between that of the granulosa cell tumor variety and that of constitutional type. The other possible causes are, as a rule, readily eliminated. For example in the adrenal cases, a precocious puberty characterized by heterologous sex characters is exhibited. They show hypertrichosis but the hair growth involves not only the genitals and axillae, but usually other parts of the body, like the abdomen or extremities, with at times tufts of hair on the back. In the granulosal and constitutional cases, on the other hand, the hair is apt to be limited to the genitals and axillae, just as it is in the girl at normal puberty.

(2) In the adrenal cases there is often marked hypertrophy of the clitoris which is never seen in the constitutional or granulosa-cell tumor cases. Finally, the precocious development seen in the adrenal cases is associated in only a minority of cases with precocious menstruation, a characteristic manifestation in the other two chief varieties.

(3) The pineal tumor group, the tumor usually being a teratoma, does not concern the gynecologist, since practically all known instances have occurred in males. Furthermore, the precocious changes are probably due to involvement of certain brain areas rather than of the pineal body itself. Other causes of precocious puberty, such as tumors of the hypothalamic areas, are commonly characterized by definite cerebral symptoms far graver than the precocious sex changes.

(4) The granulosa cell tumor variety presents changes like those we might expect from the administration of large amounts of estrogen to infants or young children, and the menstrual bleeding which they show is purely estrogen-induced and not associated with ovulation. In this important respect they differ from the far more common cases of constitutional or genic type, in which ovulation does occur, precocious pregnancy being, therefore, possible. Unless one can actually palpate an enlargement in one or the other ovary, one should lean toward the diagnosis of the constitutional type.—*Mary Frances Vastine.*

SKELETAL SYSTEM

KNEPPER, PAUL A. Parachute fractures. *Surg., Gynec. & Obst.*, July, 1945, *81*, 53–55.

A survey of fractures which occurred in a regiment of paratroop infantry during a six month period of intensive training is presented. The over-all incidence of casualties was as high as 10 per cent in some of the jumps at the beginning of training. The number of accidents was rapidly reduced until at the end of the pe-

riod of training, the casualty rate was less than 1 per cent.

The optimum speed of the airplane during release of parachutes is about 100 miles per hour for a static line jump. The jumps were made at 800 feet above ground level in the beginning of training, gradually dropping to 600 feet as the program went on. The impact at which the parachutist hits the ground is equal to that of a fall from a height of 10 to 15 feet. A wind of over twelve to fifteen miles per hour greatly increases the number of casualties.

Types of Injuries in Parachute Jumpers.

1. Psychoneuroses, including hysterical states and malingering, were very uncommon.

2. Back injuries were not common. There were 7 cases of compression fractures of the vertebrae. None was complicated by paralysis. No cases of protruded intervertebral disc were found. Back sprains of any severity were uncommon as the men were taught to land on their feet and roll forward. The 7 cases of compression fracture were all associated with one or two complications; the primary "chute" did not open and the secondary "chute" was used, or another parachutist accidentally emptied part of the air from the "chute" which carried the man who was injured. There were no compound fractures and no fatal accidents.

3. Head injuries included 2 linear fractures and 4 basilar ones. These injuries all happened in a high wind which caused the parachutist to land sideways, striking the head in some manner.

4. Acromioclavicular separations were not common. Injuries to the acromioclavicular joint were treated by depression of the clavicle and immobilization. Separation of the conoid and trapezoid ligaments was repaired by open operation.

5. Fracture of the posterior lip of the tibia is so typical a parachute injury that it has been named "paratrooper fracture" by Tobin. Parachutists were taught to land on the ball of the foot. This is possible if there is no wind and the terrain is even, but too often this is not the situation. These fractures occur when the weight of the body is transmitted through one foot instead of being divided.

A method of treatment was established: (1) A padded plaster cast is applied from the base of the toes to the tibial tuberosity. (2) The foot is placed in the neutral position. (3) The ankle is placed in exactly 90 degrees dorsiflexion. (4) An anesthetic may be necessary to get the ankle in proper position. (5) The patient is on absolute bed rest for one week, with elevation of the extremity on two pillows. (6) After one week, the cast is removed and replaced by another cast with a very small amount of padding. A section of wool felt 2 by 2 inches is placed dorsally over the tarsal bones. (7) After forty-eight hours the patient is allowed to walk on the leg. No crutches or walking calipers are employed. The cast will frequently break on the bottom but good support is given to the ankle nevertheless. (8) The cast is removed after two weeks and daily whirlpool baths of thirty minutes in the morning and infra-red treatment for one hour in the afternoon are given. One week of this treatment will suffice ordinarily and parachutist is ready for full duty, including marches.—*Mary Frances Vastine.*

SCHMIER, ADOLPH A. The internal epicondylar epiphysis and elbow injuries. *Surg., Gynec. & Obst.*, April, 1945, *80*, 416–421.

Fracture dislocation of the elbow in children with inclusion of the internal humeral epicondyle into the joint is not a frequent injury. It is not as rare, however, as was formerly believed. The injury is of extreme importance since the full significance of the pathology is not always appreciated. The dislocation is easily diagnosed, but the displacement of the internal epicondyle into the joint is often unrecognized; the dislocation is reduced but the epicondyle remains within the joint. As a result the child may be doomed to markedly restricted painful range of motion in the elbow. It is highly important to suspect and look for a displacement of the internal humeral epicondyle in every case of elbow dislocation in children.

Clinical Examination. This gives some indication of the injury present. A dislocation is obvious and is easily recognized. The normal prominence of the internal humeral epicondyle should be looked for and its absence noted. Comparison should be made with the opposite normal elbow. The presence of localized tenderness over the region of the epicondyle may be significant.

The internal humeral epicondyle gives origin to a group of muscles by a conjoined tendon. These muscles are the pronator teres, flexor carpi radialis, palmaris longus, flexor digitorum sublimis (humeral head). By their action these muscles pronate the forearm, flex the wrist and fingers, and aid in flexing the elbow.

The center of ossification of the internal humeral epicondyle appears at about the age of five years and fuses at about the age of eighteen years.

Evaluation of Study. Avulsion of the internal humeral epicondyle may be simple or may accompany elbow dislocation. Conversely, dislocation of the elbow may occur alone or be accompanied by a separation of the internal humeral epicondyle. In those cases of dislocation in which the epicondyle becomes displaced within the joint it is highly important that the entire pathology be recognized early and treated without delay. Complete reduction can be obtained in those cases that are treated early by the closed method described by the author. The maximum length of time following injury in which closed reduction may be successful cannot be stated with accuracy. All of the patients in this series in which the dislocations were successfully reduced by the closed method were treated within twenty-four hours after injury. All of them obtained well functioning painless elbows.—*Mary Frances Vastine.*

MISCELLANEOUS

Merritt, E. A., Den, A. J., and Wilcox, U. V. The effects of radiation therapy in clostridium infection in sheep. *Radiology*, Oct., 1944, *43*, 325–329.

Experiments in the treatment of clostridium infection in sheep by irradiation were carried on for four months in 1942. Sheep were selected as experimental animals because the surface irradiated is a prime factor in computing radiation doses and this surface is about the same in sheep as in man. Twelve sheep with an average weight of 80 lb. were used and later 4 sheep with an average weight of 120 lb. added. The latter strain was more resistant to the disease than the former and so modified the statistical results.

The mortality in the untreated animals of the first group was 86 per sent, while after the addition of the second group it was 75 per cent; the mortality of the treated animals in the first group was 25 per cent before including the additional animals and 29 per sent after including them. The gangrenous process was more extensive in the untreated than in the treated animals. In all the treated animals the infection was limited to the inoculated thigh and leg and never spread across the perineum or along the flank on the affected side. The immunity of animals should be tested before they are used for such experiments and those that are found immune rejected.

The regulation U. S. Army mobile field unit was used for examination and treatment. Doses from 600 to 1,600 r were given, but treatment was determined in each case by the severity of the disease and the general condition of the animal. The best results were seen in animals that were given an early initial treatment of 300 r with a second treatment of at least 200 r within six hours. There is no absolute rule for treatment except to treat early and adequately. —*Audrey G. Morgan.*

Quimby, Edith H. Dosage table for linear radium sources. *Radiology*, Dec., 1944, *43*, 572–577.

Information in regard to dosage in gamma roentgens from linear sources such as are used in the treatment of cervical and rectal cancer has not heretofore been available. The author describes her method of determining dosage along a linear source and gives a diagram illustrating it and a table showing the gamma roentgens per 100 mg-hr. delivered at various distances from various points along such linear sources. No allowance is made for tissue absorption or scatter. Examples illustrating the use of the table are given. It is a simple matter to determine the dose delivered fat any point within a reasonable range of any linear source or simple combination of such sources kept in place for any specified time by the methods illustrated in these examples.—*Audrey G. Morgan.*

Gray, L. H., Ellis, F., Fairchild, G. C., and Paterson, E. Dosage-rate in radiotherapy; symposium. *Brit. J. Radiol.*, Nov., 1944, *17*, 327–342.

This is a very technical study of the effect of dosage rate in radiotherapy, illustrated with curves and tables showing the conclusions arrived at and the reasons for such conclusions. It is based on experimental work on isolated tissues and cells, and the problems faced by the radiotherapist in treating the human body are more complex but the conclusions reached here may prove a valuable guide to the therapist.

It seems probable that greater effects can be obtained by the use of high dosage rates than by the use of low ones and that a high intensity irradiation causes more damage to malignant

tissue than a low intensity irradiation without a corresponding increase in the damage to normal tissue. This conclusion seems to be confirmed by histological study and clinical observation. It is probable that the damage to malignant tissue is as permanent with high intensity irradiation as with low intensity irradiation. It is also probable that increased effects are obtained by prolonging the total time of irradiation and that the cause of this better effect is a greater uniformity of response in the cells of irradiated cultures.—*Audrey G. Morgan.*

MAYNEORD, W. V. Energy absorption. III. The mathematical theory of integral dose and its applications in practice. *Brit. J. Radiol.*, Dec., 1944, *17*, 359–367.

This is a very technical discussion of the mathematical theory of integral dose in whole body irradiation. "Volume integral dose," "surface integral dose" and "line integral dose" are defined and the mathematical working out of the conclusions given in detail. Tables are given showing data in regard to integral dose during whole body irradiation of patients of varying size and shape. In a later paper it is hoped to discuss problems in the study of radium therapy with the sources of radiation near to or inside the body. Two mechanical methods of finding integral dose are described, one by measurement of the movement of planes about a fulcrum and the other by weighing "tongues" of isodose curves cut from the roentgen film and dividing the weight of the bundle of tongues by the weight per unit area of the film. This is perhaps the easiest method of finding surface integral dose in a given plane. —*Audrey G. Morgan.*

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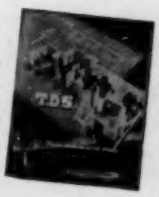

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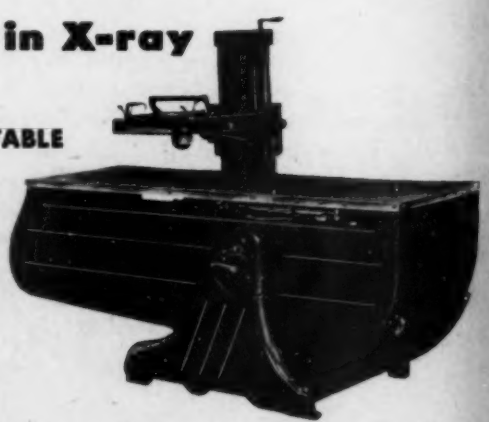

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